TRANSACTIONS

AMERICAN ACADEMY
of
OPHTHALMOLOGY
and
OTOLARYNGOLOGY

JANUARY - FEBRUARY
1953

Volume 57 Number 1

Printed bimonthly, February, April, June, August, October, and December, by the Douglas Printing Company, 109 North 18th Street, Omaha 2, Nebraska, for the American Academy of Ophthalmology and Otolaryngology. Editorial and business office, 100 First Avenue Building, Rochester, Minnesota. Subscription price $5.00 yearly to members, $10.00 yearly to non-members. Entered as Second-Class Matter November 28, 1938, at the Post Office at Omaha, Neb., under Act of March 8, 1879.

ANNUAL MEETING, OCTOBER 11-16, 1953, CHICAGO

Introducing

OpH TRADEMARK *Eye Drops*

STERILE
BUFFERED
SOLUTION

DECONGESTANT · ASTRINGENT ANTISEPTIC

CONTAINS Neo-Synephrine® hydrochloride.....0.08 (1/12)%
For Prompt and Prolonged Decongestant Action

Zinc Sulfate.....0.06%
Astringent and Antiseptic

Boric Acid.....2.2%
For Nonirritating Antisepsis

Chlorobutanol.....1 grain per fl. oz.
As preservative

OpH a dependable, soothing eye lotion for temporary relief of the congestion and discomfort due to minor ocular irritations.

SUPPLIED in unique sealed dropper-vial of 10 cc. protecting sterility

INC. NEW YORK 18, N. Y. WINDSOR, ONT.

TRANSACTIONS

AMERICAN ACADEMY *of* OPHTHALMOLOGY *and* OTOLARYNGOLOGY

VOLUME 57 JANUARY - FEBRUARY, 1953 NUMBER 1

TABLE OF CONTENTS

PAGE

REMARKS ON PATHOLOGY AND ITS ROLE IN THE PRACTICE OF OPHTHALMOLOGY AND OTOLARYNGOLOGY 7
PLINN F. MORSE, M.D., Detroit, Mich., by invitation

THE ATLASES OF PATHOLOGY 13
WILLIAM L. BENEDICT, M.D., Rochester, Minn.
BRIGADIER GENERAL GEORGE R. CALLENDER, Washington, D. C.
BRIGADIER GENERAL ELBERT DECOURSEY, Washington, D. C.
COLONEL J. E. ASH, Washington, D. C.
BRITTAIN F. PAYNE, M.D., New York, N. Y.
HELENOR CAMPBELL WILDER, Washington, D. C.
MURIEL RAUM, M.D., Washington, D. C.
JONAS S. FRIEDENWALD, M.D., Baltimore, Md.
EDMUND P. FOWLER, JR., M.D., New York, N. Y.

SOME EXPERIMENTAL OBSERVATIONS BEARING ON MENIERE'S DISEASE 27
Wherry Memorial Lecture
JOHN R. LINDSAY, M.D., Chicago, Ill.

A STUDY OF THE ANATOMY AND FUNCTION OF THE TECTORIAL MEMBRANE ... 35
A. C. HILDING, M.D., Ph.D., Duluth, Minn.

TUMORS OF THE NECK 48
LOUIS H. CLERF, M.D., Philadelphia, Pa.

PROPER USE OF ANTIMICROBIAL DRUGS IN ACUTE OTITIS MEDIA 53
MIRIAM H. RUTHERFORD, M.D., Oakland, Calif.

A STRONGER MAGNIFIER FOR ENDOSCOPES (*A New Instrument*) 62
WILLIAM T. K. BRYAN, M.D., St. Louis, Mo.

CORTISONE AND HYDROCORTISONE IN OCULAR INFECTIONS 64
PHILLIPS THYGESON, M.D., San Jose, Calif.,
MICHAEL J. HOGAN, M.D., and SAMUEL J. KIMURA, M.D.,
San Francisco, Calif.

ACCIDENTAL SURGICAL INJURY TO THE LENS 80
LEONARD CHRISTENSEN, M.D., and
EDWARD MCLEAN, M.D., by invitation, Portland, Ore.

FURTHER OBSERVATIONS ON INTRAOCULAR ACRYLIC LENSES IN CATARACT SURGERY 98
HAROLD RIDLEY, M.D. (Cambridge), London, England, by invitation

A CASE OF MULTIPLE MALIGNANT MELANOMAS IN ONE EYE 107
DAVID A. ROSEN, M.D., and
GARDNER N. MOULTON, M.D., Baltimore, Md., by invitation

NEW INSTRUMENTS

PAGE

New Scissors for Enlargement of Cataract Incision 109

Improved Motorized Instrument for Dacryocystorhinostomy and Cataract Extraction by Suction 110
Ramón Castroviejo, M.D., New York, N. Y.

Chart for Testing Near Vision 112

Superior Oblique Tucker 113
Walter H. Fink, M.D., Minneapolis, Minn.

Operating Light (Modified Ortho-Lite) 114

A Modified Cystotome to Make the Incisions for Preplaced Sutures 115
Walter S. Atkinson, M.D., Watertown, N. Y.

In Memoriam 116

American Association of Eye, Ear, Nose and Throat Society Secretaries: Annual Meeting — 1952 117

News Notes XXIX

Positions Available XXXII

Directory of Ophthalmologic and Otolaryngologic Societies XXXIII

Hearing Aids Accepted by Council on Physical Medicine and Rehabilitation of the American Medical Association XLII

Upjohn

cortisone for inflammation, neomycin for infection:

Neosone

Trademark

OPHTHALMIC OINTMENT

Each gram contains:

Cortisone Acetate . . . . . 15 mg.

Neomycin Sulfate . . . . . 5 mg.

(equivalent to 3.5 mg. neomycin base)

Available in 1 drachm tubes with applicator tip

The Upjohn Company, Kalamazoo, Michigan

FOR THOROUGH SUBJECTIVE TESTING

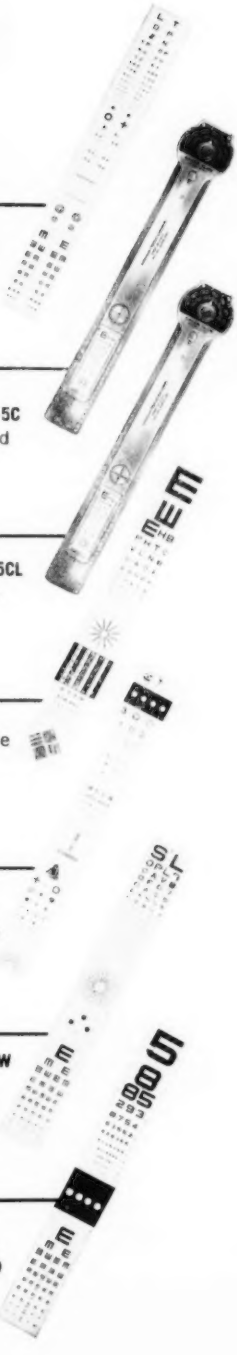
AO PROJECT-O-CHART

Slides

Table Model No. 1217-A Project-O-Chart

The AO Project-O-Chart helps you to complete a general and accurate examination within a short time—because of the speed with which you can switch from test to test. Over 20 different slides are now available including combinations of characters for general refraction, illiterates, children, and in foreign languages. For further information ask your AO Representative.

American Optical
COMPANY
INSTRUMENT DIVISION • BUFFALO 15, NEW YORK



NEW MORGAN SLIDE No. 1217-21 for monocular refraction under binocular fixation.

ROBINSON-COHEN SLIDE No. 1217-5C —all inclusive monocular and binocular subjective test.

ROBINSON-COHEN SLIDE No. 1217-5CL —same as 1217-5C, but with rotating disc composed of solid cross lines on a clear background.

CHARACTER SLIDE No. 1217-1—Wide selection from 20/400 to 20/10, astigmatic square and sunburst dial.

COMBINATION SLIDE No. 1217-2—Includes 4 color test objects, child's pictures for retinoscopy, Verhoeff test and improved bichrome test.

WORTH FOUR DOT TEST No. 1217-W —Includes characters for 20/200 to 20/10, astigmatic, and illiterate tests, plus reversible spectacle frame with red and green filters.

ILLITERATE SLIDE No. 1217-20—Includes E's from 20/200 to 20/15, numbers from 20/400 to 20/10 and simple color recognition.

Have you gotten your copy of

Ophthalmic Pathology?

The Atlas and Textbook published under the Sponsorship of the American Academy of Ophthalmology and Otolaryngology

A NEW BOOK — unlike any other in the field — that is unsurpassed in authority and usefulness. Here are some of the features:

1. *Authority* — The book was published under the sponsorship of The American Academy of Ophthalmology and Otolaryngology — together with The Armed Forces Institute of Pathology. Written by outstanding investigators and practitioners of ophthalmology, it gives you the most advanced thought available today on the pathologic aspects of the specialty.

2. *Magnificent Illustrations* — In no other volume will you find clearer or more helpful illustrations of the pathology of the eye. The 963 illustrations on 240 plates were carefully selected from a huge collection compiled over the past thirty years.

3. *Useful Text* — Not only does the text present a clear coverage of the morphologic pathology of the eye — but it also discusses the *physiologic* mechanisms that influence pathologic changes.

You'll find a great wealth of vitally helpful material in this new work. So make sure you have your copy!

By JONAS S. FRIEDENWALD, HELENOR CAMPBELL WILDER, A. EDWARD MAUMENEE, T. E. SANDERS, JOHN E. L. KEYES, MICHAEL J. HOGAN, W. C. and ELLA U. OWENS, with the editorial assistance of HELEN KNIGHT STEWARD. 500 pages, 7½" x 11", with 963 illustrations on 240 figures. $18.00. **Published under the joint sponsorship of The American Academy of Ophthalmology and Otolaryngology and The Armed Forces Institute of Pathology.** **New!**

ORDER FORM ▸

Too-1-53

W. B. SAUNDERS COMPANY
West Washington Square, Philadelphia 5

Please send me and charge my account
☐ 30 days ☐ Easy Payment Plan

OPHTHALMIC PATHOLOGY $18.00

Name _____

Address _____

BLAKISTON
the
MEDICAL
AND SCIENCE
PUBLISHERS

JUST PUBLISHED!

MELLER'S

NEW SIXTH EDITION

Ophthalmic Surgery

REVISED AND ENLARGED

by DR. J. BOCK

Head, Department of Ophthalmology, University of Gratz, Austria, and Dr. Meller's Associate at Vienna for many years

Supplement by K. Kofler

Translated and Edited by

Ray K. Daily, M.D., F.A.C.S., and Louis Daily, Jr., M.D., Ph.D.

This, the SIXTH EDITION of the German Classic by J. Meller, represents a complete revision of Meller's work by Dr. J. Bock, Dr. Meller's associate and co-author. It includes the ophthalmologic surgical procedures adopted by the Vienna Clinic since Dr. Meller's retirement. However, only the operations which have been thoroughly tried, evaluated and proved best at the Vienna Clinic are described. New material is presented which includes a comprehensive description of the operations for retinal detachment and for the removal of intraocular bodies. The outstanding features of the book are the unsurpassed and detailed descriptions of the techniques of each operative procedure. In fact, the techniques of the operations are described in such detail that they can readily be followed by surgeons with only a grasp of the FUNDAMENTALS of ophthalmology. There is also a supplement by Dr. K. Kofler on intranasal surgery of the lacrimal sac, of interest to rhinologists as well as ophthalmologists. To this new edition the translators have added an appendix, which consists of a description of procedures developed but recently.

507 Pages; 287 Illustrations..... **12.00**

ORDER HERE

THE BLAKISTON COMPANY, INC.

575 Madison Avenue
New York 22, New York

105 Bond Street
Toronto 2, Canada

Please send me_____copies of OPHTHALMIC SURGERY,
New Sixth Edition by J. Meller.....$12.00

☐ Charge ☐ Check or M. O. Enclosed ☐ Send C.O.D.

NAME_____

ADDRESS_____

CITY_____ ZONE_____ STATE_____

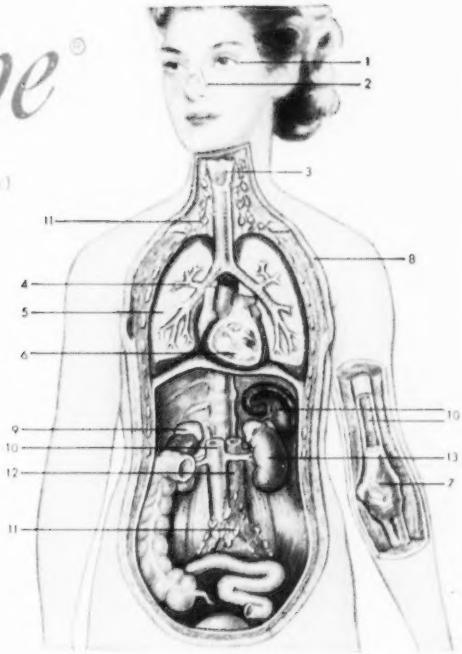
TAAOO 2/53

Cortone®

ACETATE

(CORTISONE ACETATE, MERCK)

The many indications for CORTONE *highlight its therapeutic importance in everyday practice*



Primary Site of Pathology and Indications

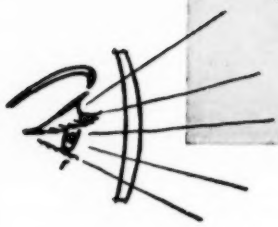
1. EYE—Inflammatory eye disease. **2. NOSE**—Intractable hay fever. **3. LARYNX**—Laryngeal edema (allergic). **4. BRONCHI**—Intractable bronchial asthma. **5. LUNG**—Sarcoidosis. **6. HEART**—Acute rheumatic fever with carditis. **7. BONES AND JOINTS**—Rheumatoid arthritis; Rheumatoid spondylitis; Acute gouty arthritis; Still's Disease; Psoriatic arthritis. **8. SKIN AND CONNECTIVE TISSUE**—Pemphigus; Exfoliative dermatitis; Atopic dermatitis; Disseminated lupus erythematosus; Scleroderma (early); Dermatomyositis; Poison Ivy. **9. ADRENAL GLAND**—Congenital adrenal hyperplasia; Addison's Disease; Adrenalectomy for hypertension, Cushing's Syndrome, and neoplastic diseases. **10. BLOOD, BONE MARROW, AND SPLEEN**—Allergic purpura; Acute leukemia† (lymphocytic or granulocytic); Chronic lymphatic leukemia.† **11. LYMPH NODES**—Lymphosarcoma†; Hodgkin's Disease†. **12. ARTERIES AND CONNECTIVE TISSUE**—Periarteritis nodosa (early). **13. KIDNEY**—Nephrotic Syndrome, without uremia (to induce withdrawal diuresis). **14. VARIOUS TISSUES**—Sarcoidosis; Angioneurotic edema; Drug sensitization; Serum sickness; Waterhouse-Friderichsen Syndrome.

†Transient beneficial effects.

CORTONE is the registered trade-mark of Merck & Co., Inc. for its brand of cortisone.

MERCK & CO., INC.
Manufacturing Chemists
RAHWAY, NEW JERSEY

Orthogon Lenses are made the way they are because eyes work the way they do



The image formed by any off-center portion of a lens is degraded by the amount of astigmatic error introduced. This aberration, known as marginal or oblique astigmatism, is one for which the eye can make no compensation or accommodation. And this is the aberration for which the Orthogon series is specifically corrected. Before you prescribe any "corrected" lens, be sure you know *what* it's corrected for. Bausch & Lomb Orthogon is corrected to eliminate the effect of marginal astigmatism—it makes possible clear center-to-edge vision.

In Soft-Lite, too

The Orthogon system of lens correction is based on the principle that only through reduction of astigmatic variation ... to a level below the physiological limits of human perception ... can utmost clarity of vision be achieved.

BAUSCH & LOMB Orthogon LENSES

AVAILABLE IN THE WIDEST RANGE OF SINGLE-VISION,

ABSORBTIVE AND MULTI-FOCAL TYPES

1853/1953 INTO A NEW CENTURY OF OPTICAL PROGRESS

BAUSCH & LOMB CENTENNIAL

Acclaimed by Reviewers Everywhere!

Reese's TUMORS OF THE EYE

By

ALGERNON B. REESE

Attending Ophthalmologist and Pathologist, Institute of Ophthalmology, Presbyterian Hospital; Ophthalmologist to Memorial Center for Cancer and Allied Diseases; Clinical Professor of Ophthalmology, College of Physicians and Surgeons, Columbia University, New York

584 pages, 511 illus., 122 in full color, $20.00

EXCERPTS FROM A FEW OF THE ENTHUSIASTIC REVIEWS!

● "Occasionally, but only occasionally, a really good book appears in medicine; this is one."
— STEWART DUKE-ELDER IN BRIT. M. J.

● "The years of experience of the author in the clinic and pathologic laboratory, the meticulous care reflected on every page, his scientific integrity, assure an accuracy of text that will be difficult to equal and impossible to surpass."
— DERRICK VAIL IN AM. J. OPHTH.

● "This book is a comprehensive and authoritative treatise, which will be of very great value. . . . Dr. Reese has combined a detailed account of the pathology of ocular tumors with an analysis of their clinical course richly illuminated by his own vast experience and embellished with sagacious comments in respect to differential diagnosis, surgical techniques, and other therapeutic procedures. The whole is set forth in a fluent and eminently readable text. The illustrations are excellent."
— ARCH. OPHTH.

● "Exceedingly practical volume for any specialist who may encounter tumors of the eye, its adnexa and their often far reaching complications."
— BERTHA A. KLIEN IN SURG., GYNEC. & OBST.

● ". . . more than five hundred illustrations are superb in their explanation of the text . . . a more complete and comprehensive group cannot be found anywhere."
— DIGEST OPHTH. & OTOLARYNG.

You May Order This Valuable Book on Approval

ON APPROVAL ORDER COUPON

PAUL B. HOEBER, Inc.
Medical Book Department of Harper & Brothers
49 East 33rd Street, New York 16, N. Y.

Send me ON APPROVAL:
Reese's TUMORS OF THE EYE . . . $20.00

☐ Check Enclosed ☐ Charge My Account

NAME

ADDRESS

CITY ZONE STATE

TR 253

JAMES HOWARD KINDELBERGER

Aviation executive of international renown; distinguished engineer and designer; chairman of the Board and chief executive officer of North American Aviation, Inc.; served as Air Officer, World War I; one of America's most expert and sought-after counselors in all phases of the aviation industry.

ERNST FREDERICK WERNER ALEXANDERSON

World-famous electrical engineer; one of America's outstanding inventors; has obtained more than 300 U. S. patents; recipient of international medals, awards, honors; noted pioneer in radio, television, electric ship propulsion, railroad electrification; consulting engineer for General Electric Company.

CHARLES EARL DAVY

Director of Engineering Operations and member of the Engineering Board of Chrysler Corporation; associated with advanced engineering designs and procurement of vital material; prominent in World War II aircraft and tank engine programs; identified with top industry projects for more than thirty years.

WILLIAM C. EDDY, CAPTAIN USN (RET.)

Famed pioneer in all branches of television engineering; holder of numerous audio-visual patents; instrumental in building the Navy's World War II radar training program; cartoonist and author; respected leader in research and development projects; President and active head of Television Associates, Inc.

These four great American engineering executives, who have a thorough understanding of electrical and mechanical devices, can afford any type of hearing aid at any price. They wear the seventy-five dollar Zenith hearing aid.

BIOGRAPHICAL DATA FROM FILES OF "WHO'S WHO IN AMERICA."

Terramycin

CRYSTALLINE

OPHTHALMIC

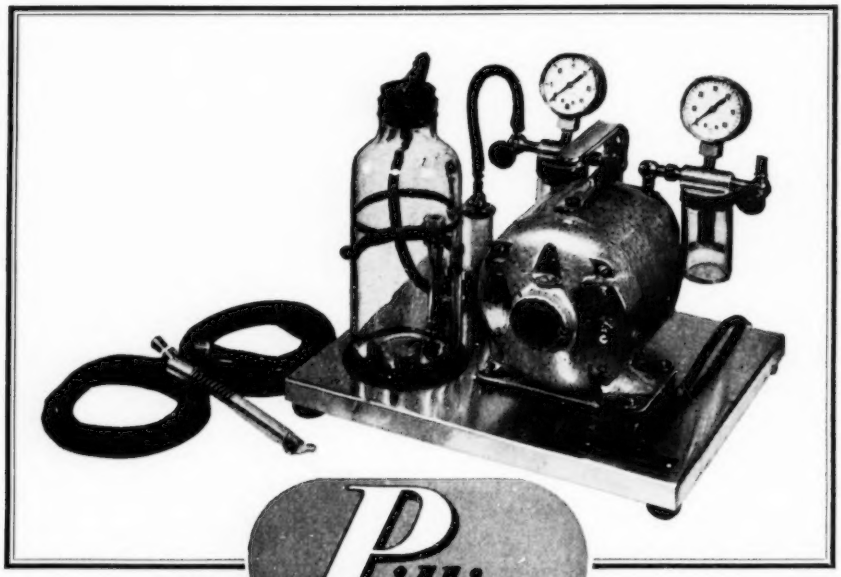
Indicated in a wide range of external ocular infections involving diverse structures and tissues of the eye, Terramycin Ophthalmic preparations are effective and valuable either as the sole medication or as an adjunct to oral Terramycin therapy. It is only in the rare case that the use of Terramycin Ophthalmic Ointment or Solution is attended by sensitizing reactions.

Supplied: CRYSTALLINE TERRAMYCIN HYDROCHLORIDE OPHTHALMIC OINTMENT, *5 mg. per Gm. ointment; tubes of* $\frac{1}{8}$ *oz.*

CRYSTALLINE TERRAMYCIN HYDROCHLORIDE OPHTHALMIC SOLUTION, *5 cc. vials containing 25 mg. for preparation of topical solutions isotonic with lacrimal fluid and buffered to pH 8.2.*

Terramycin is also available as *Capsules, Tablets, Oral Suspension, Oral Drops* and other convenient dosage forms.

ANTIBIOTIC DIVISION Pfizer CHAS. PFIZER & CO., INC., *Brooklyn 6, N.Y.*



The Pilling

PORTABLE PUMP

FOR SUCTION AND PRESSURE

- Easy to operate—simple to control
- Large, easy-to-read suction and pressure gauges
- Readily accessible regulating valves
- Completely portable, yet stays firmly in position while in use

The sturdiest and most useful pump of its size available—at a price that cannot be matched.

The Pilling Portable Pump will give yeoman's service all through the hospital. It is ideal for office, hospital bedside and even house-call use . . . easily carried wherever it's needed—no trouble at all to maintain.

Supplied complete with suction and pressure hoses. 110 volts, 60 cycles, AC.

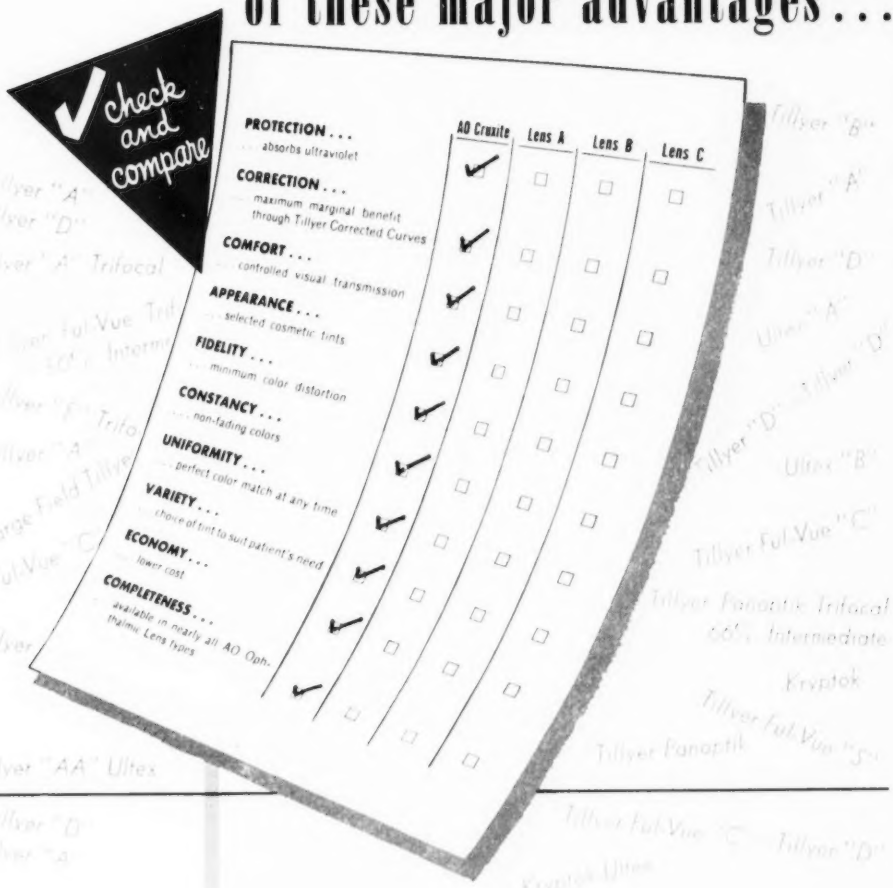
Only **$99.50** f.o.b. Philadelphia

Order direct from

GEORGE P. Pilling & SON CO.
3451 WALNUT STREET PHILADELPHIA

A Standing Invitation: *When in Philadelphia, visit our new salesrooms. Free parking for doctors in our private lot.*

Only AO TILLYER CRUXITE provides ALL of these major advantages...



Available in a wide range of Single Vision, Bifocal and Trifocal Lens Types . . . many with AO Tillyer Curves.

Compare these advantages of AO Tillyer Cruxite with the features of *any* other light absorptive lens.

Some lenses have several of these characteristics . . . others have different ones.

But ONLY AO Tillyer Cruxite provides ALL of them!

Even with these advantages, AO Tillyer Cruxite is still *less* expensive than many major competitive light absorptive lenses.

American Optical

OPHTHALMIC LENS DIVISION

The Monel Metal

RADIUM

NASOPHARYNGEAL APPLICATOR

ACTUAL LENGTH, 17 CM.

for the treatment of hyperplastic lymphoid tissue at the orifice of the eustachian tube or in the fossa of Rosenmuller.

The Radium Chemical Company originated this type applicator and has supplied more than 1500 to Otolaryngologists in the United States.

Available through purchase or annual lease.

WRITE FOR COMPLETE DETAILS . . . NO OBLIGATION

RADIUM CHEMICAL CO., INC.

161 East Forty-Second St., New York 17, N. Y.

MORE THAN 35 YEARS CONTINUOUS SERVICE TO THE MEDICAL PROFESSION

When Clinical Proof is Your Guide

DOHO RESEARCH PRODUCTS

are indicated . . .

Now !!!

The NEW O-TOS-MO-SAN

is a *Specific* in Suppurative Ear Infections — both Acute and Chronic, also External Otitis because it is . . .

BACTERICIDAL . . . **(GRAM-POSITIVE — GRAM-NEGATIVE) — it KILLS BACTERIA, including BACILLUS PROTEUS, B. PYOCYANEUS, E. COLI, BETA HEMOLYTIC STAPHYLOCOCCUS AUREUS**
(Isolated from ear infections and found resistant to antibiotics in laboratory tests)

and

FUNGICIDAL . . . **it KILLS FUNGI — including ASPERGILLI, TRICOPHYTON, MONILIA, and MICROSPORUM**

NON-TOXIC • NON-IRRITATING
STABLE • CLEAR

PROVED EFFECTIVE AGAINST ANTIBIOTIC RESISTANT STRAINS OF ORGANISMS
Substantiating Laboratory and Clinical data in press.

FORMULA:
A NEW, improved process, using Doho glycerol base, results in a chemical combination having these valuable properties.

Urea	2.0 GRAMS
Sulfathiazole	1.6 GRAMS
Glycerol (DOHO) Base	16.4 GRAMS

(Highest obtainable spec. grav.)

Challenge !!!

TRY NEW O-TOS-MO-SAN in your *most stubborn cases*, the results will prove convincing.

DOHO CHEMICAL CORP., 100 Varick Street., New York 13, N. Y.

AURALGAN — After 40 years STILL the auralgesic and decongestant • **RHINALGAN — safe nasal decongestant. Acts locally NOT systemically.**

THE HOUSE IRRIGATOR

A simple non-drip irrigator, useful for fenestration, brain surgery, etc., or any place where a small volume at low pressure is required. Slight pressure on the rubber body of the irrigator causes the solution to flow and automatically shuts off when this pressure is released. Can be sterilized or autoclaved. Can be repaired instantly in the office. No moving metal parts. Described in the Archives of Otolaryngology, October 1951.

Price $6.50

Surgical Mechanical Research
1905 Beverly Blvd.
Los Angeles 4, Calif.

THE NEW YORK POLYCLINIC
MEDICAL SCHOOL AND HOSPITAL
(ORGANIZED 1881)
(The Pioneer Post-Graduate Medical Institution in America)
EYE, EAR, NOSE AND THROAT

A combined full-time course covering an academic year (9 months). It consists of attendance at clinics; witnessing operations; lectures; demonstration of cases and cadaver demonstrations; operative eye, ear, nose and throat (cadaver); head and neck dissection (cadaver); clinical and cadaver demonstrations in bronchoscopy, laryngeal surgery and surgery for facial palsy; refraction; radiology; pathology, bacteriology and embryology; physiology; neuro-anatomy; anesthesia; physical medicine; allergy; examination of patients pre-operatively and follow-up post-operatively in the wards and clinics. Also refresher courses (3 months); attendance at departmental and general conferences.

For information about this course address
THE DEAN: 345 West 50th St., NEW YORK 19, N. Y.

THE MAKING AND FITTING OF ARTIFICIAL EYES ARE A SPECIALTY WITH US . . .

Not a Sideline!

We have a complete selection service for glass and plastic eyes. Within 8 hours after receiving your order, we send you a selection of eyes on memorandum. Write for further information and free color chart and size guide.

CHICAGO
DETROIT
CLEVELAND
KANSAS CITY
MINNEAPOLIS
NEW ORLEANS
ST. LOUIS

SERVING THE PROFESSION
SINCE 1851

NEW YORK
BALTIMORE
BOSTON
BUFFALO
PHILADELPHIA
PITTSBURGH
WASHINGTON

MAGER & GOUGELMAN, INC.

Jewelled HEIRESS

Gold Lace HEIRESS

Petite Gold Lace HEIRESS

HEIRESS does wonderful things for her and for you

The moment she puts on an Heiress frame—she adds that inimitable touch of richness and exquisite styling to her wardrobe that can only come with *all-gold.* For whether it is jewelled, gold lace, petite gold lace, plain or engraved—she can be sure that Heiress compliments her good taste and marks her a person of discrimination.

You, too, will find a prestige builder in this superb frame.

NEW JERSEY OPTICAL COMPANY · IRVINGTON, N.J.

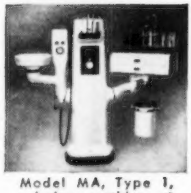
WITH A *Ritter* ENT UNIT

As a busy physician you can conserve your energy, yet serve more patients with a Ritter ENT Unit . . . designed especially to help you utilize your skills more thoroughly. You can treat patients without moving from the chair. A stretch of the arm brings air, water, vacuum, electricity, or waste into immediate use. Equally accessible are spray bottles, medicaments and low voltage instruments. Diagnostic and treatment time is kept to a minimum . . . with patients more at ease. Low voltage instruments are properly angled for easy grasp.

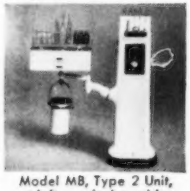
Then, too, there is a Ritter ENT Unit to fit your favorite operating technique. The Ritter cuspidor can be on the right or left as part of the unit, or, as a separate piece of equipment. Ritter ENT Units are made to position at either right or left of the chair.

Start now to enjoy the advantages of a Ritter ENT Unit best suited to your technique.

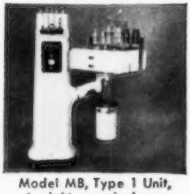
Choose the UNIT to fit your technique



Model MA, Type 1, swinging cuspidor at right of chair.



Model MB, Type 2 Unit, at left, surgical cuspidor at right of chair.



Model MB, Type 1 Unit, at right, surgical cuspidor at left of chair.

FOR ADVANCED EQUIPMENT LOOK TO

Ritter

COMPANY INCORPORATED
RITTER PARK, ROCHESTER 3, N. Y.

restore free breathing...

In the common cold, rhinitis syndromes, and hay fever 'Vasoxyl' Intranasal quickly dispels hyperemia and edema of the nasal mucosa, thus restoring patency of the passages and making for good ventilation.

'Vasoxyl'® Intranasal

brand

METHOXAMINE HYDROCHLORIDE 0.25% SOLUTION

- *is not irritating, even to infants;*
- *is most unlikely to give rise to secondary engorgement;*
- *does not inhibit cilia;*
- *does not cause central stimulation or sleeplessness.*

Bottles of 1 fl. oz., with separate dropper, and 1 pint

Burroughs Wellcome & Co. (U.S.A.) Inc., *Tuckahoe 7, N. Y.*

Doctor, be your own judge... try this simple test

With so many claims made in cigarette advertising, you, Doctor, no doubt prefer to judge for yourself. So won't you make this simple test?

Take a PHILIP MORRIS and any other cigarette

1. Light up either one first. Take a puff—get a good mouthful of smoke—and s-l-o-w-l-y let the smoke come *directly* through your nose.
2. Now, do exactly the same thing with the other cigarette.

Notice that PHILIP MORRIS is definitely less irritating, definitely milder.

PHILIP MORRIS

Philip Morris & Co. Ltd., Inc., 100 Park Avenue, New York 17, N. Y.

SYMPOSIUM: CORNEAL DISEASES

Moderator: Frederick C. Cordes, M.D.

PANEL:

David G. Cogan, M.D.
James H. Allen, M.D.
F. Phinizy Calhoun, Jr., M.D.
Edwin B. Dunphy, M.D.
Kenneth C. Swan, M.D.

We have a number of reprints of this excellent symposium, which appeared in the March-April 1951 TRANSACTIONS.

While they last,

$0.25 per copy

Send order with remittance to
W. L. BENEDICT, M.D.
100 First Avenue Building
Rochester, Minn.

New Color Slide
LIBRARIES OF THE EYE

from the private collections of recognized experts

Series
I. Anterior Segment Photographs
II. Anterior Segment Photographs
III. Retinal Color Photographs
IV. Gonioscopic Color Photographs
All by H. Saul Sugar, M.D., Detroit
V. Retinal Color Photographs
from the collection of the late Robert Von Der Heydt, M.D., Chicago
VI. Original Ophthalmological Photomicrographic Slides
by Julius Weber, New York

Write for booklet listing each slide. Literature also available on lighting systems and complete camera units for photographing the eye.

BARNETT Optical Laboratories
2419 N. Halsted St., Chicago 14, Ill.

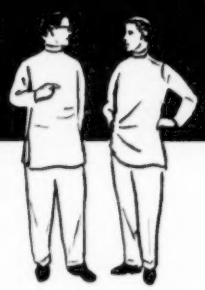
COMPLETE OPHTHALMIC SERVICE FOR THE PROFESSION
Since 1913

Benson
OPTICAL COMPANY

MAIN OFFICE AND LABORATORY • MINNEAPOLIS, MINN.

Branch Laboratories in Principal Cities of Upper Midwest

"I am extremely pleased with your product"



MONOCAINE

LOCAL ANESTHETIC

IN METAL CAP ANESTUBES

"I am extremely pleased with your product. In tonsillectomies performed under local anesthesia, it has reduced my anesthesia time by 50 per cent ...and the amount of operative discomfort to the patient, to a minimum."

DR. C. H. R.

This comment is typical of users of Monocaine for local anesthesia. Monocaine has been used in tens of millions of injections in all types of minor surgery. It produces deep, profound, uneventful anesthesia, without side reactions. Return to normal is rapid. The Metal Cap Anestube system of administration permits direct injection from the cartridge into tissues and eliminates the possibility of syringe breakage or leakage.

41 Years Against Pain SINCE 1911

NOVOCOL CHEMICAL MFG. CO., INC.
2911-23 Atlantic Ave., Brooklyn 7, N. Y

Please send detailed information on Monocaine Local Anesthetic and its convenient administration in Metal Cap Anestubes.

Dr. .....

Address .....

.....

NOVOCOL CHEMICAL MFG. CO., INC.

2911-23 Atlantic Ave., Brooklyn 7, N. Y.

Toronto • London • Buenos Aires • Rio de Janeiro

. . . . *for Suspension Laryngoscopy*

Invaluable

equipment for direct examination—including biopsy—of all structures, through the wide open mouth. . . . No part of the instrument lies in the field of examination. . . . The surgeon's hands are left entirely free to make the most careful examination.

SE-850 Killian Suspension Gallows. Clamps onto the table to hold the Lynch hook in the desired operating position. Adjusts smoothly in every necessary direction, and will not slip. Easily and quickly taken down for compact storage or carrying. Each, **$145.00**

SE-860 Lynch Suspension Hook. Completely adjustable. Spring clips prevent accidental disengagement from the gallows arm. Complete with five sizes of spatulae, and right and left tooth plates. Black plated finish is non-reflecting. Each, **$145.00**

Instrument Makers to the Profession Since 1895

V. Mueller and Company

330 S. HONORE STREET CHICAGO 12, ILLINOIS

THE "LACRILENS"® IS A SAFE LENS

1. Free from Danger of Injury to the Eyes.
2. Will Not Fall Out of the Eye.
3. Safe for All Sports, Including Swimming.
4. Custom Made to Fit the Eye.

Write for Brochure

Obrig Laboratories Inc.

49 EAST 51st STREET • NEW YORK, 22, N. Y.

Manufacturers of all types of Contact Lenses

BRANCHES IN

PHILADELPHIA . . . . . MONTREAL . . . . . LONDON

JOHANNESBURG . . . SHANGHAI

®

New ACADEMY *Publications*

GLAUCOMA

A Manual by SAMUEL J. MEYER, M.D.

$1.00

RETINAL DETACHMENT

H. ARRUGA, M.D., et al.

With Color Plates

$1.00

Send order with remittance to

W. L. BENEDICT, M.D.

100 First Avenue Building **ROCHESTER, MINN.**

Piromen*

(BACTERIAL POLYSACCHARIDE)

for the effective control of a wide variety of ALLERGIES and DERMATOSES

Every day more physicians are discovering the early clinical benefits effected by the administration of ***Piromen***, employed either as a specific, or concomitantly with other drugs.

Piromen is a biologically-active bacterial polysaccharide which produces a marked leucocytosis and a stimulation of the reticulo-endothelial system. It is nonprotein, nonantigenic, and may be employed safely within a wide range of dosage.

Piromen is prepared in stable colloidal dispersion for parenteral use. It is supplied in 10 cc. vials containing either 4 gamma (micrograms) per cc., or 10 gamma per cc.

For a comprehensive booklet detailing the use of this new therapeutic agent, merely write "***Piromen***" on your *Rx* and mail to—

Manufactured by

TRAVENOL LABORATORIES, INC.

Subsidiary of BAXTER LABORATORIES, INC., MORTON GROVE, ILLINOIS

*Trade Mark

CAMEL

Mild, Flavorful

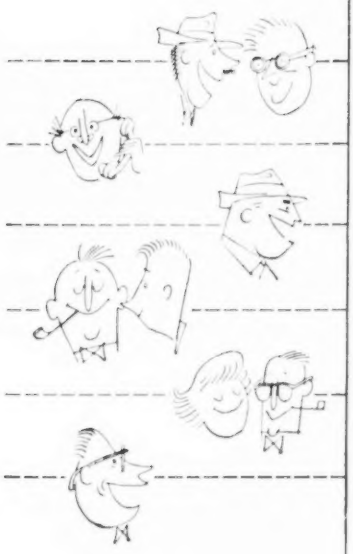
pack after pack!

AMERICA'S MOST POPULAR CIGARETTE

WHAT THEY'RE SAYING ABOUT THE NEW

Shuron®

RONSIR BROWLINE FRAME...



"It's certainly a winner"

"The best man's frame in the world"

"Perfect for men"

"A hit if I ever saw one"

"Best reaction I've ever had"

"Men go for it"

The Ronsir is only one of the Shuron Browline Family of Fine Frames that are available from Independent Suppliers.

Shuron®

SHURON OPTICAL COMPANY, INC.

GENEVA, N. Y., *Established 1864*

AVAILABLE SUPPLIES

MANUALS

	Price
OCULAR SURGERY, Conrad Berens and Michel Loutfallah	$1.00
RETINAL DETACHMENT, H. Arruga et al.	1.00
CATARACT TYPES, F. C. Cordes	1.00
SLIT LAMP BIOMICROSCOPY, E. L. Goar et al	1.00
OUTLINE OF NEURO-OPHTHALMOLOGY, P. J. Leinfelder	1.00
A COMPREHENSIVE DESCRIPTION OF THE ORBIT, ORBITAL CONTENT, AND ASSOCIATED STRUCTURES WITH CLINICAL APPLICATIONS, R. E. McCotter et al	1.00
GLAUCOMA, Samuel J. Meyer	1.00
THE INTERPRETATION OF VISUAL FIELDS (Revised), C. W. Rucker	1.00
DISTURBANCES OF OCULAR MOTILITY, Richard G. Scobee	1.00
REFRACTION, Daniel Snydacker and Frank W. Newell	1.00
EXTRINSIC EYE MUSCLES, H. Saul Sugar	1.00
ENDOGENOUS UVEITIS, A. C. Woods	1.00
PRINCIPLES OF OTOLARYNGOLOGIC PLASTIC SURGERY, Oscar J. Becker	1.00
RADIOGRAPHIC ANATOMY OF THE TEMPORAL BONE, J. Brown Farrior	1.00
NONSURGICAL COMPLICATIONS OF OTOLARYNGOLOGIC SURGERY, J. J. Pressman et al	1.00

MONOGRAPHS

HELPFUL HINTS FOR HANDLING THE HEARING HANDICAPPED, Gordon D. Hoople et al	$0.25
HEARING TESTS, Arthur L. Juers	0.25
THE EMBRYOLOGY OF THE EAR, NOSE, AND THROAT, O. E. Van Alyea	0.25
CORNEAL DISEASES: A SYMPOSIUM, Frederick C. Cordes et al	0.25
REFRACTION DIFFICULTIES (Revised) Clarence A. Veasey, Jr.	0.50

ABSTRACTS

Each set contains the available outlines and briefs of instruction courses as presented at Academy meetings. Unbound.

Ophthalmology:	1937, 1938, 1942, 1948 @	1.50
	1949	3.50
	1950, 1951, 1952	5.00
Otolaryngology:	1937, 1938, 1939, 1941, 1942 @	1.50
	1949	3.50
	1950, 1951, 1952	4.00

TRANSACTIONS

Bound Volumes:	Record of meetings held in: 1917-18, 1919, 1920, 1921, 1923, 1924, 1925, 1926, 1927, 1928, 1930, 1932, 1933, 1934, 1935, 1936, 1937, 1938, 1939, 1940, 1941, 1942, 1943 @	$1.50
	1949-50 bound volume	5.00
	1950-51 bound volume	5.00
	1952 bound volume	5.00
Subscription:	Per year, bimonthly publication	10.00
Single issue:	Before 1950	0.50
	January-February 1950—November-December 1952, per copy	1.00
	Issues of 1953, per copy	2.00

SEND ORDER WITH REMITTANCE TO

W. L. Benedict, M.D.
100 First Avenue Building
Rochester, Minnesota

TRANSACTIONS

AMERICAN ACADEMY
of
OPHTHALMOLOGY
and
OTOLARYNGOLOGY

VOLUME 57
1953

PRINTED IN U.S.A.

OFFICERS *for* 1953

President
FREDERICK C. CORDES

Executive Secretary-Treasurer
WILLIAM L. BENEDICT

First Vice-President
THOMAS C. GALLOWAY

Second Vice-President
HARVEY E. THORPE

Third Vice-President
HENRY B. ORTON

Secretary for Ophthalmology
KENNETH L. ROPER

Secretary for Otolaryngology
HOWARD P. HOUSE

Secretary for Instruction
Ophthalmology
A. D. RUEDEMANN

Secretary for Instruction
Otolaryngology
and Maxillofacial Surgery
DEAN M. LIERLE

Secretary for Home Study Courses
LAWRENCE R. BOIES

Secretary for Public Relations
ERLING W. HANSEN

THE COUNCIL — 1953

American Academy of Ophthalmology and Otolaryngology

Frederick C. Cordes, San Francisco, Calif. ..... President
† John J. Shea, Memphis, Tenn. ..... President-Elect
Thomas C. Galloway, Evanston, Ill. ..... First Vice-President
Harvey E. Thorpe, Pittsburgh, Pa. ..... Second Vice-President
Henry B. Orton, Newark, N. J. ..... Third Vice-President
J. Mackenzie Brown, Los Angeles, Calif. ..... Past President
Derrick Vail, Chicago, Ill. ..... Past President
James Milton Robb, Detroit, Mich. ..... Past President
William L. Benedict, Rochester, Minn. .. Executive Secretary-Treasurer / Editor and Business Manager of the TRANSACTIONS
Kenneth L. Roper, Chicago, Ill. ..... Secretary for Ophthalmology
Howard P. House, Los Angeles, Calif. ..... Secretary for Otolaryngology
A. D. Ruedemann, Detroit, Mich. ..... Secretary for Instruction in Ophthalmology
Dean M. Lierle, Iowa City, Iowa ..... Secretary for Instruction in Otolaryngology and Maxillofacial Surgery
Lawrence R. Boies, Minneapolis, Minn. ..... Secretary for Home Study Courses
Erling W. Hansen, Minneapolis, Minn. ..... Secretary for Public Relations
C. Stewart Nash, Rochester, N. Y. ..... Councillor
John H. Dunnington, New York, N. Y. ..... Councillor
Francis E. LeJeune, New Orleans, La. ..... Councillor
S. Rodman Irvine, Beverly Hills, Calif. ..... Councillor

COUNCIL COMMITTEES

EXECUTIVE, RESEARCH AND FINANCE

Frederick C. Cordes, *Chairman*
J. Mackenzie Brown — † John J. Shea
Derrick Vail — C. Stewart Nash
James Milton Robb — William L. Benedict

RESEARCH AND FINANCE

(Sub-Committee of Executive, Research and Finance)
J. Mackenzie Brown, *Chairman*
Frederick C. Cordes — William L. Benedict

ACTIVITIES

C. Stewart Nash, *Chairman*
John H. Dunnington — † John J. Shea
Francis E. LeJeune — Thomas C. Galloway
S. Rodman Irvine

JUDICIAL

Frederick C. Cordes, *Chairman*
C. Stewart Nash — Francis E. LeJeune
John H. Dunnington — S. Rodman Irvine

BOARD OF COUNCILLORS

C. Stewart Nash, *Chairman*
John H. Dunnington — S. Rodman Irvine
Francis E. LeJeune

BOARD OF SECRETARIES

William L. Benedict, *Chairman*
Kenneth L. Roper — Dean M. Lierle
Howard P. House — Lawrence R. Boies
A. D. Ruedemann — Erling W. Hansen

PROGRAM

Frederick C. Cordes, *Chairman*
† John J. Shea — Howard P. House
Thomas C. Galloway — A. D. Ruedemann
C. Stewart Nash — Dean M. Lierle
William L. Benedict — Lawrence R. Boies
Kenneth L. Roper — Erling W. Hansen

STANDING COMMITTEES

ARMED FORCES

M. Elliott Randolph, M.D., *Chairman*, Baltimore, Md.
Gordon M. Bruce, M.D. — New York, N. Y.
Arthur M. Culler, M.D. — Columbus, Ohio
Gordon D. Hoople, M.D. — Syracuse, N. Y.
Frank D. Lathrop, M.D. — Boston, Mass.
Brittain F. Payne, M.D. — New York, N. Y.
Harry P. Schenck, M.D. — Philadelphia, Pa.

CONSERVATION OF HEARING

Executive Committee
Dean M. Lierle, M.D., *Chairman*, Iowa City, Iowa
Lawrence R. Boies, M.D. — Minneapolis, Minn.
Norton Canfield, M.D. — New Haven, Conn.
Kenneth M. Day, M.D. — Pittsburgh, Pa.
Aram Glorig, Jr., M.D. — Washington, D. C.
Gordon D. Hoople, M.D. — Syracuse, N. Y.
Howard P. House, M.D. — Los Angeles, Calif.
Charles E. Kinney, M.D. — Cleveland, Ohio
W. J. McNally, M.D. — Montreal, Que., Canada
C. Stewart Nash, M.D. — Rochester, N. Y.
George E. Shambaugh, Jr., M.D. — Chicago, Ill.
Henry L. Williams, M.D. — Rochester, Minn.

† Deceased

STANDING COMMITTEES — *Continued*

Senior Counselors

Gordon Berry, M.D.	Worcester, Mass.
George M. Coates, M.D.	Philadelphia, Pa.
Carl H. McCaskey, M.D.	Indianapolis, Ind.
Harris P. Mosher, M.D.	Marblehead, Mass.

Consultants

Raymond Carhart, Ph.D.	Evanston, Ill.
Eva Thompson Carson	Glenview, Ill.
Howard A. Carter	Chicago, Ill.
Hallowell Davis, M.D.	St. Louis, Mo.
Stacy Guild, Ph.D.	Baltimore, Md.
Scott N. Reger, Ph.D.	Iowa City, Iowa

Ophthalmic Pathology

Michael J. Hogan, M.D., *Chairman*, San Francisco, Calif.

F. Phinizy Calhoun, Jr., M.D.	Atlanta, Ga.
John S. McGavic, M.D.	Bryn Mawr, Pa.
Theodore E. Sanders, M.D.	St. Louis, Mo.

Otolaryngic Pathology

Paul H. Holinger, M.D., *Chairman*,	Chicago, Ill.
David D. DeWeese, M.D.	Portland, Ore.
Edmund P. Fowler, Jr., M.D.	New York, N. Y.
John R. Lindsay, M.D.	Chicago, Ill.
James W. McLaurin, M.D.	Baton Rouge, La.
Lewis F. Morrison, M.D.	San Francisco, Calif.
John A. Murtagh, M.D.	Hanover, N. H.

Pan-American Relations in Ophthalmology

Conrad Berens, M.D., *Chairman* New York, N. Y.

Pan-American Relations in Otolaryngology

C. L. Jackson, M.D., *Chairman*	Philadelphia, Pa.
Victor R. Alfaro, M.D.	Washington, D. C.
Norton Canfield, M.D.	New Haven, Conn.
John Marquis Converse, M.D.	New York, N. Y.
Paul H. Holinger, M.D.	Chicago, Ill.
Howard P. House, M.D.	Los Angeles, Calif.
LeRoy A. Schall, M.D.	Boston, Mass.
Theodore E. Walsh, M.D.	St. Louis, Mo.

Plastic Surgery

John Marquis Converse, M.D., *Chairman*, New York, N. Y.

Wendell L. Hughes, M.D., *Co-Chairman*, New York, N. Y.

Alston Callahan, M.D.	Birmingham, Ala.
William C. Huffman, M.D.	Iowa City, Iowa
Dean M. Lierle, M.D.	Iowa City, Iowa
A. D. Ruedeman, M.D.	Detroit, Mich.
Byron Smith, M.D.	New York, N. Y.
Edmund B. Spaeth, M.D.	Philadelphia, Pa.

Prevention of Blindness

Ralph I. Lloyd, M.D., *Chairman*	Brooklyn, N. Y.
Lawrence T. Post, M.D.	St. Louis, Mo.
Alan C. Woods, M.D.	Baltimore, Md.

Scientific Exhibit

Kenneth L. Roper, M.D., *Chairman* Chicago, Ill.
Eugene L. Derlacki, M.D., *Co-Chairman*, Chicago, Ill.

Standardization of Tonometers

Peter C. Kronfeld, M.D., *Chairman*	Chicago, Ill.
Moacyr E. Alvaro, M.D.	Sao Paulo, Brazil
Jonas S. Friedenwald, M.D.	Baltimore, Md.
W. Morton Grant, M.D.	Boston, Mass.
Trygve Gundersen, M.D.	Boston, Mass.
D. O. Harrington, M.D.	San Francisco, Calif.
Thomas H. Hodgson, M.D.	Toronto, Ont., Canada
John C. McCulloch, M.D.	Toronto, Ont., Canada
John M. McLean, M.D.	New York, N. Y.
Adolph Posner, M.D.	New York, N. Y.
Robert N. Shaffer, M.D.	San Francisco, Calif.

Study of Audio-Visual Instruction

Dean M. Lierle, M.D., *Chairman*, Iowa City, Iowa
A. L. Benedict, M.D., *ex officio*, Rochester, Minn.

Howard P. House, M.D.	Los Angeles, Calif.
Kenneth L. Roper, M.D.	Chicago, Ill.
A. D. Ruedemann, M.D.	Detroit, Mich.

Teachers' Section

Otolaryngology

Lawrence R. Boies, M.D., *Chairman*, Minneapolis, Minn.

Gordon D. Hoople, M.D.	Syracuse, N. Y.
Percy E. Ireland, M.D.	Toronto, Ont., Canada
Dean M. Lierle, M.D.	Iowa City, Iowa
James H. Maxwell, M.D.	Ann Arbor, Mich.
Lewis F. Morrison, M.D.	San Francisco, Calif.
LeRoy A. Schall, M.D.	Boston, Mass.
Theodore E. Walsh, M.D.	St. Louis, Mo.

Ophthalmology

A. D. Ruedemann, M.D., *Chairman*, Detroit, Mich.

Frederick C. Cordes, M.D.	San Francisco, Calif.
John H. Dunnington, M.D.	New York, N. Y.
Edwin B. Dunphy, M.D.	Boston, Mass.
Lawrence T. Post, M.D.	St. Louis, Mo.
Edmund B. Spaeth, M.D.	Philadelphia, Pa.
Kenneth C. Swan, M.D.	Portland, Ore.
Derrick Vail, M.D.	Chicago, Ill.

Representatives of the Academy to:

American Association for the Advancement of Science

M. Elliott Randolph, M.D. Baltimore, Md.

American Board of Ophthalmology

Derrick T. Vail, M.D.	Chicago, Ill.
F. Bruce Fralick, M.D.	Ann Arbor, Mich.
Gordon M. Bruce, M.D.	New York, N. Y.
Alson E. Braley, M.D.	Iowa City, Iowa

American Board of Otolaryngology

Francis E. LeJeune, M.D.	New Orleans, La.
Gordon F. Parkhurst, M.D.	Davenport, Iowa

American College of Surgeons

Board of Governors

James H. Maxwell, M.D.	Ann Arbor, Mich.
Carl H. McCaskey, M.D.	Indianapolis, Ind.
Conrad Berens, M.D.	New York, N. Y.

American Committee on Optics and Visual Physiology

Alfred Cowan, M.D.	Philadelphia, Pa.
Kenneth C. Swan, M.D.	Portland, Ore.
Frederick C. Cordes, M.D.	San Francisco, Calif.

American Orthoptic Council

Kenneth C. Swan, M.D. Portland, Ore.
William Egbert Krewson, III, M.D., Philadelphia, Pa.
Frank D. Costenbader, M.D. Washington, D. C.

Joint Committee on Industrial Ophthalmology

Edmund B. Spaeth, M.D.	Philadelphia, Pa.
John B. Hitz, M.D.	Milwaukee, Wis.
Col. Victor A. Byrnes, M.C., Randolph Field, Texas	

Joint Committee on Retrolental Fibroplasia

William C. Owens, M.D. Baltimore, Md.

National Society for Crippled Children and Adults, Inc.

Dean M. Lierle, M.D. Iowa City, Iowa

REMARKS ON PATHOLOGY AND ITS ROLE IN THE PRACTICE OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

PLINN F. MORSE, M.D.
DETROIT, MICH.

BY INVITATION

WHEN a hospital pathologist is asked to address this most distinguished group of physicians at the joint meeting of the two Sections of your Academy, he must approach his task with misgiving.

I take it from the Chairman's introductory remarks that the Council would like to stimulate your interest in pathology as it relates to your two fields of specialization. This is undoubtedly a healthy sign, and a general auditing of your group attitude toward pathology will be helpful if it can be accomplished.

With the characteristic pragmatic approach of practicing physicians whose first concern is the care of the sick, you have already gone a long way in the accomplishment of this effort by the publication of your two atlases illustrating particular lesions in your respective fields. From this beginning we all feel sure that works of great interest and educational value will result. It is particularly fortunate that the format of these two books makes it implicit that the effort will continue and the volumes expand into a shape never quite finished, but far more complete and valuable to the practitioner than anything heretofore attempted.

In my judgment your action has been very much more sensible than the establishment of a so-called registry. I have watched many "registries" come into being and fade into oblivion. They all tend to become mere archives of pathological specimens, interesting and valuable to the curator but not of any wide teaching value for the use and benefit of the practicing physician. The atlases possess all the advantages of a registry with the added quality of being generally useful to those who need them most and whose interest in pathology needs most to be stimulated.

Personally I am particularly pleased that the effort has been cooperative from the beginning and that the contributions come from your own members and from widely separated geographical sources.

If I may be allowed to add a thought or two aimed at future development of these volumes, it occurs to me that the consultative suggestions of one or more general pathologists of wide experience might be helpful. I say "add" with a considered purpose because I was taught early in life that the function of a consultant, if he is to be popular with the family doctor, is to "add" all the diagnoses he pleases but never to subtract any already expressed to the family if he expects to be called in on the next case.

The intensive development of the pathology of special tissues by clinical experts in various specialties has been valuable in many instances, as witness the contributions to neuropathology and bone pathology by physicians not primarily trained as general pathologists, but I think that all general pathologists feel that these subjects would have been still further advanced by the collaboration of one or more workers in the realm of general pathology. There is too great a tendency to attach local names to le-

Presented at the Fifty-Seventh Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 12-17, 1952, Chicago, Ill.

sions whose nosological position in the general scheme is obvious to the general pathologist, such for example as the word "meningioma."

Furthermore, the general pathologist will usually resist the use of terms which are substitutes for more accurate descriptive diagnoses of lesions, such as lymphoblastoma and neuroblastoma. Fortunately we have not yet begun to talk of laryngoblastoma, rhinoblastoma and tonsilloblastoma.

Also as the volumes expand, additional emphasis could be placed on the relationship of the lesions in your field to general systemic diseases.

We all feel that this plan to stimulate interest and knowledge in the field of pathology among the members of your group, although a sound objective, is difficult to accomplish. To practice your specialties successfully requires most careful time scheduling of your working hours. Your office practice is large and you see many patients in a day. Your operative schedules are difficult to arrange to cover your daily requirements, and in your field especially it is necessary that you see ninety-eight cases posing no new problem or offering no stimulating angle of interest or investigative effort in order that you may see two that are worthy of your more protracted attention.

I am reminded of a verse from Ecclesiastes which Dr. Klemperer has used on occasion, "For a dream cometh through the multitude of business."

I speak to you with great humility because there are many sitting in this room who individually know much more about the pathology of the eye, ear, nose and throat than I will ever know. The only possible service I can perform is to broaden your interest a little and try to sell the project to those of you who may be doubting Thomases.

I do not concede that these atlases are most useful to the aspiring resident who expects to take his Board examinations. They are valuable for that purpose, but if that were the only reason for their publication, I doubt that the effort would be justified. The oldest, the most experienced, and the busiest practitioner among you will be benefited most.

I have observed for many years that those busy surgeons who have made a practice personally of seeing everything that goes through the pathological laboratory, and who are always present at clinical and pathological conferences, no matter what the subject for discussion in the whole wide field of medicine and surgery, are the ones at the top of their departments and the ones of national prominence. The old saying, "if you want something done, give the job to a busy man," applies here. Those who have the least time to waste are the ones most interested in the field of pathology in any large hospital.

It is often asserted that the specialty of ear, nose and throat is a contracting field due to the introduction of antibiotics. It is true that these weapons have made some operations less often necessary, but this factor is more than offset by the many new procedures that are coming into prominence and by the very great, if belated, appreciation of the fact that many patients suffering from systemic diseases have occasion first to consult the ophthalmologist or otolaryngologist. Another fact that has become apparent to me over the years is that the lesions you have to cope with are progressively more serious and require greater skill in management. Patients turned away as hopeless a few years ago are now treated with great skill and success. The patients who come to your department in a large hospital are on the average afflicted with more serious and difficult complaints. As Dr. Robb has often mentioned to me, he has a much larger proportion of seriously ill patients to care for than in days gone by.

If I possessed the skill to picture for you the progress of the study of disease down through the years and were able to show you the many facets of a crystal still growing by deposits of the now invisible particles in the solution of cosmic knowledge, I would feel that I had accomplished my assignment.

We are accustomed to dwell too much on the attainments of the present and to discount the great accomplishments of the past.

Let us not pass too lightly or with scorn over the philosophical and mystical notions in which our guild had its beginnings. Medicine like astronomy and all science starts with folklore, superstition and mysticism of a kind that often amazes us at its accuracy or near accuracy.

When the pre-Hippocratic and Hippocratic physician spoke of blood, phlegm, yellow bile and black bile, did he not have a prescient knowledge of things to come?

We pretend to have given over and abandoned the old humoral pathology. The concept that blood which comes from the heart represents heat; phlegm from the brain, cold; yellow bile secreted by the liver, dryness, and black bile from the spleen and stomach, wetness.

When we remember the poverty of the age in natural science we are not moved to scorn these ideas as mystical nonsense, but we are given pause to reflect that the experienced physician of that day, who had seen as many sick people as you or I, instinctively and dimly sensed that there were such things as hydration, dehydration and heat regulation, and when he said "pneuma" he was quite as close to the mark as many a physician of today who talks about oxidation and anoxemia without bothering to understand thoroughly the already known physiological phenomena involved. What I am trying to say is that the philosopher of old with his "pneuma" sensed something instinctively and was more honest and more scientific than many a modern physician who talks glibly of principles that he does not understand. The difference between the expert and the mediocre in our profession is that the expert is not too proud to study and investigate those phenomena not in his immediate field that will benefit his practice and his patients!

An example comes to mind. I happen to know that a nationally prominent member of your Academy is at present making a thorough review of the physical chemistry of hydrogen-ion concentration and only laid the subject down to come to this meeting.

If I could stimulate those of you to whom the subject matter is new to go back to the sources, even superficially, so that you might get a perspective of our science, your interest in pathology would be assured.

Physicians are like politicians: they swing from one extreme to the other. We gave up and anathematized the humoral pathology and crases of Hippocrates down through Rokitansky and then embraced the philosophy of electrolyte exchange, endocrine integration of cellular function, the Selyé alarm reaction and the pituitary and other dystrophies.

Our humoral pathology of yesterday is our biochemistry of today, and our revilement of crasis that, except for his great mental balance, almost discouraged Rokitansky from producing the final edition of his great descriptive pathology, has changed to hymns of praise with new titles such as the lymphatic constitution, endocrine and hereditary dystrophies and many others. How would Rokitansky have reacted to Dr. Klemperer's work on the collagen diseases! I think they would not have had much difference of opinion.

Verily history repeats itself. One hundred years ago Virchow prefaced his lectures on the cellular pathology with these words:

> In consequence of the great advances that have been made in the details of science, it has been becoming continually more and more difficult to the majority of those who are engaged in practice, to obtain in the subjects treated on in these lectures that amount of personal experience which alone can guarantee a certain degree of accuracy of judgment. Day by day do those who are obliged to consume their best energies in the frequently so toilsome and so exhausting routine of practice find it becoming less and less possible for them, not only closely to examine, but even to understand the more recent medical works.
>
> In a science so directly practical as that of medicine, and at a time when such a rapid accumulation of facts is taking place, as there is in ours, we are doubly bound to render our knowledge accessible to the whole body of our professional brethren. Let us undertake, therefore, every one of us to fulfil the duties both of an observer and of an instructor.
>
> Those, who have found leisure enough to keep up their knowledge by reading the current medical literature, will find but little that is new in these lectures. The rest will not, by reading them, be spared the trouble of being obliged to study the subjects, which are here only briefly touched upon, more closely in the histological, physiological and pathological works.

No better preface to your two atlases could possibly have been written. And now, one hundred years later we can find no better way to express the responsibilities of the busy practitioner toward the growing science of medicine.

A few paragraphs ago I spoke of the study of disease down through the years. Members of your Academy should be particularly interested and impressed with the important role that ophthalmology and otolaryngology have played in the advance of pathology. Remember that pathology is the great grandchild of clinical medicine. There was no professorship of pathology in any university until 1819, when Lobstein was made professor in an independent new department of Pathology in the University of Strasbourg at the suggestion of the great biologist, Cuvier. All the advances in general and descriptive pathology up to that time had been the work of clinicians. The accessibility of the eye, ear, nose and throat to examination made it natural that those regions were concerned with many of the early descriptions. Quickly let us recall just a very few. Of the many biblical references so often repeated I am much interested in two especially. One presages our modern knowledge of vitamin deficiency of particular concern to the ophthalmologist.

"And the wild asses did stand in the high places, they snuffed up the wind like dragons: *their eyes did fail because there was no grass.*"—Jeremiah 14:6.

Xerophthalmia, a vitamin deficiency disease, is not a discovery of our generation. A striking example referring to general pathology is found in I Samuel 6:5. After a description of the plague that overwhelmed the Philistines while they were in possession of the Ark of the Covenant, we read: "Wherefore ye shall make images of your emerods and *images of your mice* that mar the land."

The folk knowledge of the importance of rodents in the spread of plague goes back at least to Babylonian times.

Hippocratic medicine concerned itself with nasal catarrh, laryngitis and pneumonia. Tubercles were the result and not the cause of pulmonary hemorrhage. Middle ear disease was a complication of brain abscess until Morgagni put the sequence to rights, although his preceptor Valsalva had believed the Hippocratic teaching. It is amazing that Morgagni should have described the cataract that bears his name, considering the tools with which he worked.

The list already grows too long, but let us quickly pass over Helmholtz' in-

vention of the ophthalmoscope. This was an interesting switch of events in that a physiologist-pathologist-physicist invented something useful.

As Ophthalmology and Otolaryngology were emerging as clinical subjects in their own right, enormous contributions were being made by general surgeons. We can mention only a few. Joseph Lister, who the pathologists like to remind you was basically trained in microscopic anatomy, wrote on the structure of the iris and its action in dilating the pupil. Versatility was the order of the day. Ferdinand von Grafe of Warsaw wrote on Cesarean section, rhinoplasty and cleft palate.

Viktor von Bruns about 1850 wrote on the surgery of the larynx.

Maunoir of Geneva: construction of an artificial pupil and surgery of cystic tumors.

The first tonsillotome was invented by the father of American surgery, Philip Syng Physick.

As we review the progress that pathology has made from the earliest recorded observations we are impressed by two obvious features of the record.

From the first the advances in our knowledge have been characterized by periods of productivity followed by periods of silence. These periods of silence have often lasted several hundred years. Viewing the state of the world at each period of progress or desuetude we find that the political and economic picture of the times corresponds closely to the activity of scientific thought.

More important than this, however, and especially important to us as present-day physicians is the fact that the great advances were made by men who were either primarily clinicians or who had from the beginning the clinical point of view and approach.

Two examples of this thought will suffice.

From the time of Anthony Benevieni in Florence writing in 1507 down to Morgagni in 1767, a period of 260 years, there were numerous works on pathological anatomy including the remarkable "Sepulchretum" of Bonnet in 1697, all of which fell on deaf ears. The reason, it seems to me, is that they lacked or were not in accord with the clinical point of view.

The element that made Morgagni's work great was that his reports included the clinical course and case history in addition to the descriptive anatomical findings, and thus physicians began to interpret clinical findings in terms of the abnormalities they saw at the autopsy table.

Not to take any honor or reverence away from Rokitansky for his great work on anatomical pathology, a work which Virchow, his most cutting critic, admitted had never been equalled for its lucid excellence of description, we can see clearly from this distance that the weakness of the work was Rokitansky's absorption in the appearances found in the dead body without having sufficient concern with the clinical phenomena the patient had presented during his sickness.

Read if you will Rokitansky's chapter on the pathology of the pituitary. He had in his thousands of autopsies seen most if not all of the lesions of this structure. He differentiated the lesions of the anterior lobe, pars intermedia, infundibulum and posterior lobe; but with the dead body before him he says not a word that makes us think that he was aware of the relation of the hypophysis to the several dystrophies we recognize now as a matter of course. He could not, with his vast experience, have escaped making these discoveries if he had had an interest in the patient's sickness during life or if he had had the case history before him. At times he comes close to seeing some of the rela-

tionships. He mentions the coexistence of hypoplasia of the adrenals and generalized lymphoid hyperplasia. He had the material at hand, if he had only known it, to defend his doctrine of crases.

This blind spot in his presentation is all the more remarkable for the reason that in his survey of the historical and contemporary progress in pathology up to the time of his publication, Rokitansky mentions mostly clinicians as having placed the foundation stones of the subject.

We should be continually alert against repeating this failure, of which we are all guilty, the failure to correlate the pathological findings with the clinical phenomena.

To illustrate this I have a personal experience to recount which is most embarrassing. Many years ago, before much was known of the physiology and pathology of the parathyroid, I had occasion to study at autopsy a case of osteitis fibrosa cystica. This case had been thoroughly studied in the wards by all the then known methods, including x-rays of the skeleton as well as metabolic and calcium balance studies in the laboratory. At autopsy I found a large adenoma of a parathyroid gland and recorded it in the protocol without any thought whatever that it had any relation to the patient's systemic disease. Years later when Mandl and Hunter and Max Ballin published their investigations, Dr. William Gordon, who had been in charge of the patient, came to me and reminded me that this patient on whom we had performed an autopsy years before had had a parathyroid adenoma. This had made so little impression on me that I had forgotten it entirely. We got out the record, the protocol and the slides, and there it was for all of us to read. I had missed entirely the significance of the autopsy finding.

As a corollary to the fact that before the specialties of ophthalmology and otolaryngology had taken shape the foundations of the subjects had been laid down by general surgeons and internists, I had intended to emphasize the fact that nowadays many general systemic diseases are first diagnosed by members of your Academy as you make your routine examinations.

To help me in this effort I asked Dr. Davis in ophthalmology and Dr. Gordon in internal medicine to furnish me with notes on the subject. The notes they furnished me were so extensive and covered so wide a field that I realized that this material should be handled as a separate subject—it is so extensive and voluminous, and covers such a wide field.

Their notes made it plain, although I myself had talked of these things before our local eye, ear, nose and throat society many years ago, that it was absolutely imperative that the ophthalmologist and otolaryngologist be informed and keep abreast of the general field of medicine and surgery. This interest, as I said a while back, is a *sine qua non* of the best qualified members of your special societies.

And now what of new diseases? If Virchow said one hundred years ago that clinicians should keep abreast of the medical literature, it is all the more imperative today, and I refer to general medical literature and not to your specialty publications. During what is rapidly becoming a long life in medicine I have seen many new diseases come into being, diseases which were as yet not recorded when I went to school. Many of these, of course, were merely examples of the belated recognition of conditions which had been before our eyes since medicine was young, but to me there can be no question that really new diseases sometimes confront us. I cannot believe that such diseases as

toxoplasmosis and histoplasmosis were not relatively new entities at the time of their discovery. Whether the parasites were new or whether a virulent mutation of an old unknown nonparasitic strain occurred, I do not know. I cannot think that we missed lethargic encephalitis for a couple of generations in our own experience, only to have it become so common and well known in the twenties.

There are many other examples that will occur to all of you, and I think we must conclude that creation is still going on and that in the future we can expect to face new diseases as yet not only undiscovered but as yet nonexistent. We do not have Hamlet's choice to "rather bear the ills we have than fly to others that we know not of." Those ills will fly to us unbidden, unwanted and unheralded.

Recent work with the electron microscope suggests to me that some large molecules of the nucleoprotein group are sometimes, by means we do not know and may never understand, endowed with the mysterious attributes of life.

From what other sort of stuff do viruses have birth?

The ghosts of these as yet unknown diseases take shape as we peer into the mists of the future and we again say with Hamlet as the ghost appeared before him, "Angels and Ministers of Grace defend us."

THE ATLASES OF PATHOLOGY

WILLIAM L. BENEDICT, M.D., Rochester, Minn.: The program that we have to present this morning marks an epoch of more than forty years of effort on the part of the Academy to integrate pathology and histopathology with the clinical curriculum of our specialties. In the minutes of the Council meetings there are recorded remarks and conversations regarding the value of pathology to our specialties by men who were teaching at that time in our larger medical schools and who realized the importance of histopathology in the clinical work of our specialties. At that time it was realized that there were very few pathologists who were well versed in the histopathology of diseases of the eye, ear, nose and throat.

Inasmuch as the histopathology of lesions of the ear, nose and throat more nearly simulated the histopathology of lesions in other parts of the body, it was not so difficult to have satisfactory pathological diagnosis made in many laboratories. But for pathology of eye specimens there were only two or three laboratories that were generally conceded to be well equipped for this sort of work.

By joint action of the American Ophthalmological Society, the Academy and the Section on Ophthalmology of the A.M.A. it was proposed to stimulate this activity. It was proposed by the academy in 1912, now forty years ago, that the dues be increased so that a sum could be obtained to establish a National Pathological Laboratory, maintained and supported by the Academy. That proposal was discussed during the next five or six years, and finally a solution was arrived at in 1921.

At an interim meeting of the Council in June 1921 "Dr. (Harry) Gradle reported on a Pathological Museum in Ophthalmology and Otolaryngology in conjunction with the Army Medical Museum." "On motion by Dr. (Lee M.) Francis, Dr. Gradle was appointed chairman of the Committee and authorized to

Presented at the Joint Session of the Fifty-Seventh Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 12-17, 1952, Chicago, Ill.

make the necessary arrangements to establish this branch of the Academy's activities." "The Council authorized the expenditure of not more than One Hundred Dollars ($100.00) of the Academy funds to defray the necessary expenses incident to the launching of this Committee's work."

"Dr. Ira Frank was appointed by the Chairman to represent the Section in Oto-Laryngology." On July 1921, "Dr. Gradle offered his report on the newly created Section on Pathology." "On motion duly seconded, the new department is designated the Section on Ophthalmic and Oto-Laryngic Pathology."

At the same meeting there was read a "Report of the Committee on Graduate Courses. Dr. Wherry, the secretary of the Committee, made a comprehensive report and asked for instructions as to the best name for this section." "Dr. Gradle moved the department be called the Section on Instruction." The motion carried.

In his verbal report to the Council, Dr. Gradle said that there had been effected a union between the American Academy and the Army Medical Museum, with the hopes of establishing a National Museum of Ophthalmic and Oto-Laryngic Pathology.

The wisdom of that procedure is now apparent, for all of the objectives envisioned by the Council have been accomplished with the results we are celebrating today.

The work of many persons has made possible the collections of slides of pathological material for study by students and particularly candidates for examination by the specialty Boards and the publication of the Pathologic Atlases.

The Fellows of this Academy are deeply grateful and appreciative of the time, effort and skill that has made possible this magnificent work. We wish to take this occasion to show our appreciation to as many of them as we can. We have, therefore, invited a number of those who have been intimately identified with the development of the Tumor Registry and the Atlases to be present here today and receive the thanks of our membership for their generous contribution because this is the year of the completion of the fourth edition of the atlases and marks a new step, not a final step, in the development of this work that was started so many years ago.

Among the men who were in the Council at that time was Dr. Harris P. Mosher, who gave from his laboratory more than six hundred specimens, which established the nucleus of the Museum of Otolaryngic Pathology. We had hoped to have Dr. Mosher here because he was a member of the Council at that time and was largely instrumental in promoting the Academy's work. We are sorry that illness has made it impossible for him to speak this morning, for we wish to express our great appreciation of his efforts and his guidance in the development of this movement.

We do, however, have with us several representatives of the Army Medical Museum, and among those with whom Dr. Gradle talked first was, I believe, the then Director or Curator of the Museum, Brigadier General George R. Callender, of the Army Medical Museum and the Registry of Pathology. May I present General Callender?

BRIGADIER GENERAL GEORGE R. CALLENDER, Washington, D.C.: Doctor Benedict has covered the early history of the pioneer activity of the American Academy of Ophthalmology and Otolaryngology in establishing what is now the American Registry of Pathology of the Armed Forces Institute of Pathology much better than I could have done. The Army Medical Museum, established in 1862 to process the pathological material of the Civil War was rejuvenated by receiving an enormous amount of

material from World War I. The staff of 1920 decided the institution should become a live activity in pathology in addition to its function of collecting, studying, and reporting on the injuries and diseases of armed conflict. Major General Merritte Weber Ireland, then Surgeon General, ably supported the decision and enabled the Museum to become an Institute of Pathology rather than a "pickle factory" as he had facetiously called it.

During the transition period in which a follow-up program was established for tumors and for determining the residual pathology of diseases of military importance, Doctor Harry Gradle approached me with his proposal to establish a registry first of Opthalmic Pathology with Otolaryngic Pathology to follow. It was a great inspiration to us to have this opportunity to cooperate with the Academy. The Registry of Ophthalmic Pathology definitely established in 1922 received material with gradually increasing frequency. Other registries were established and before 1930 when the American Registry of Pathology was established at the Army Medical Museum under the auspices of the National Academy of Sciences the success of the program was assured. Sets of slides for circulation had been made with the backing and financial assistance of the Academy which has continued with results which seem to me little less than remarkable. The present status of the Registry can be given to you by others who are to follow me, better than I possibly could.

I would like to pay tribute to the pathologists who taught the staff Ophthalmic Pathology, in which at the start we were profoundly ignorant. Doctor Verhoeff, Doctor Finoff and Doctor Feingold formed the first group and had a difficult task. Doctor Friedenwald, Doctor Theobald and others have joined Doctor Verhoeff in continuing the program.

Helenor Campbell Wilder who joined the Museum Staff shortly before the establishment of the Ophthalmic Pathology Registry and became a student of eye pathology with me and successive curators of the museum is and has been the person to whom the highest credit is due for the operation of the Registry and the success it has achieved. Her recent findings of nematode larvae in pseudoglioma hitherto blamed on almost everything but parasites, and Toxoplasma in a disease we have for many years called tuberculous indicates that progress has not ceased.

Also I would express deep appreciation to the membership of the Academy who have furnished material in ever-increasing amounts. I would also credit Doctor E. C. Ellett for being the largest single contributor and active supporter during the first ten years of the Registry's existence.

I feel personally grateful to the Academy for starting this cooperative activity with civilian medicine. In my opinion it has done more than any one thing to establish the Armed Forces Institute of Pathology in its present position and I have the personal feeling from talking with many of you that you have not been losers by your initiation and support of this activity.

Brigadier General Elbert DeCoursey, Washington, D. C.: It is always a pleasure to speak to you ophthalmologists and otolaryngologists, particularly to speak about the registries that for so many years have linked the Academy and the Armed Forces Institute of Pathology. It was in the early 30's that General Callender, Colonel Ash and General Dart were instrumental in introducing me to the registries which you were so largely responsible for founding.

To me the evolution of the pathology

registries stands out as the most important organizational development in American pathology. You are the people who had the foresight to start them. Not only have you aided—or perhaps I should say pushed—the progress of pathology, but also you have helped to broaden the horizons of pathology for the Armed Forces by your individual contributions.

The American Registry of Pathology, now part of the Armed Forces Institute of Pathology, has grown until it includes twenty-one registries. I will not attempt to name them all. The scope of some of them is defined by anatomical areas, for example, the Bladder Tumor Registry, that of others by specialties, as the Orthopedic Pathology Registry, and then there are those limited to a disease, as the Leprosy Registry. Each of the 21 registries is sponsored by one or more national medical organizations like your Academy.

These registries are an intimate part of the whole Armed Forces Institute of Pathology, not the exclusive concern of a single department or specialty. Without the cooperation and participation of all departments we could not establish or operate the registries nor could we prepare the atlases and other teaching aids.

What are the registries for? One purpose they serve is education. Here is material from world-wide sources so selected and indexed that it is readily accessible for study. This might be called education for the individual. The sets of microscopic slides made up from these collections are available on loan to those who find it more convenient to carry on their study elsewhere. For group education there are sets of lantern slides based on the most representative of the study material in various fields. Medical schools often lack tissues needed in the teaching of pathology, and it is the rare specimens that you and other specialists contribute that permit us to supply them from our collections.

One of the largest educational projects, and one that has been carried on for a number of years, is in connection with the American Society of Clinical Pathologists which is now in session in this city of Chicago. On Friday there will be a seminar—it is an annual event—and this year the subject is gynecologic disease. Dr. Arthur Hertig, Professor of Pathology at Harvard Medical School, will conduct the seminar. Almost 1,000 pathologists are enrolled and every one of them was supplied with a set of 25 slides several weeks in advance of the meeting. Where does all this material come from? It comes mostly from the Armed Forces Institute of Pathology where the 25,000 slides needed for the seminar were prepared. Again we can say that it is the contribution of material from such groups as yours that makes a project of this magnitude possible. In last year's seminar on hematopoietic pathology some most interesting specimens were from otolaryngologists.

The registry material furnishes a rich background, not only for your Atlases, but also for other textbooks and many articles. In addition to those written by members of the staff or consultants, you will note in many publications an AFIP accession number identifying the source of the illustrations, or perhaps an AMM negative number from the old Army Medical Museum, or an AIP number from the intermediate Army Institute of Pathology.

The other important function of the registry is research. The service in pathologic consultation is supported by an active follow-up section, and the data on such a large number of similar cases form the basis for valuable statistical analyses. Establishment of a registry indicates that many physicians are interested in the problems of a particular field. Let me tell you of a regis-

try that is about to be started—that of Geographic Pathology. There is already a section in the Pathology Department devoted to its study. Research in this registry will be directed toward the regional variations of disease. For example, tuberculosis in Mongolians is different from that of Europeans or from tuberculosis in South America. Why? We do not know the answer, but interest in this branch is world wide as attested to by the existence of the International Society of Geographic Pathologists. Material for the registry is accumulating as specimens demonstrating the characteristics of a disease as it occurs in various parts of the world are sent to us for comparative study. It is inevitable that many studies will involve several registries. An investigation of leprosy of the eye will bring in the Registries of Ophthalmic Pathology and Leprosy, and when we wish to determine why leprosy in the eyes of Japanese and Chinese is different from leprosy in the eyes of Filipinos, in whom it is very rare, the Registry of Geographic Pathology will also participate.

In the old building that we now occupy there are few places to study, but there are quantities of material. If you want to see a hundred cases of unusual or rare diseases, we probably have them. In 1954 we will be in our new building where there will be special study rooms for you. It is a bombproof building and these rooms will be so quiet that even if an A-bomb explodes you will be undisturbed. Although we will be able to make you more comfortable in the new building, we will welcome you just as warmly in the old, so if you have a subject you wish to investigate, write to us and we will have the material ready when you come.

Colonel J. E. Ash, Washington, D. C.: This meeting reminds me so much of my first contact with the Academy because it is so different. I do not recall the title of the particular group that I had to appear before in Atlantic City in 1937, but I do remember that Dr. Mosher was a very prominent member of that group. The Eye Registry had prospered during the previous 15 years and it had its own sets of loan slides. The Registry of Otolaryngic Pathology had rather dragged its feet and had not accumulated many cases and had no teaching material. I was warned by the then Major Dart, my predecessor as Curator, that the first job I would have when I took over the Museum in early 1937 would be the development of loan sets of slides on otolaryngic pathology. That was the purpose of my visit with the group in Atlantic City. The Academy had been contributing financial support to the Eye Registry, and I thought I might be able to get something for the Ear, Nose and Throat Registry that would help in the preparation of the new sets and in the publication of atlases of ophthalmic and otolaryngic pathology.

The atmosphere was just a bit tense. I would not say unfriendly, but there was a certain amount of skepticism in the air. In fact, it was so thick that even though my lips were parched and there were cold water and glasses on a nearby table no one offered me a drink! And I remember so distinctly Dr. Mosher asking me, "And what do *you* expect to get out of this?" I think that even back in those days our conservative Boston colleague was a little fearful of Washington bureaucracy.

I can now answer Dr. Mosher's question. It was not very long after that meeting that we began to get the most wholehearted and helpful cooperation not only from Dr. Mosher but from everybody else in the Academy. All of us who have been connected with the Academy's activities are very proud that we have had the opportunity to help in the pioneer educational program

of the Academy. It has been an inspiration to us and to our Institute.

Our original atlases were rather primitive. We had no printing facilities. The first editions of both were illustrated by actual photographs and the text was mimeographed. There were about 100 copies of each. I remember so well the job it was to collate the pages. We had the pages in a series of pigeonholes on the balcony of the old Museum and it was the habit of the few of us who were then at the Museum to stop and collate a book or two on our way back from the rest rooms on the first floor.

To appreciate the part that Dr. Mosher played in this early phase not only of the Registry, but of the Otolaryngic Atlas, one has but to thumb through that original Atlas. You will see that practically every other case was "contributed by Dr. H. P. Mosher of Boston." He continued to be a very enthusiastic friend and helper in the activity.

Through the years we have had the close cooperation of many of the Academy members — Joe Beck, Novak, Schall, Fenton, Semenov, and more recently, Dr. Fowler.

After this initial effort of using photographs we did manage to acquire a little offset reproducing machine with which the second edition was run off. While this was somewhat larger than the first, the case method of presentation was used. All the time, however, we had in mind atlases that were more comprehensive professionally and technically less amateurish. New material has been coming to hand continually since the last editions, but the busy war years at the Institute delayed its final assembly.

Dr. Raum and I are very much embarrassed that the E.N.T. Atlas is not finished, but there are sample plates and the text at our booth that will give you some idea of its contents when it finally does come from the publisher in the next few months. Of course, I blame Dr. Raum for the delay. The material, such as it was at the time was practically ready for the printer a year or so ago, but when Dr. Raum joined in the activity she scrapped most of it and began combing the great amount of material at the institute and taking new pictures. Her indefatigable industry and intelligent and meticulous attention to detail are largely responsible for the present status. The new edition really is unique, first in the scope of coverage and secondly in its presentation of the special pathology with a background of the general pathologist.

It is appropriate that General DeCoursey has placed Dr. Muriel Raum in charge of the Registry of Otolaryngic Pathology. The Academy has learned by now of the power of the woman through the success of Helenor Wilder with the Eye Registry. I prophesy that under Dr. Raum's guidance the E.N.T. Registry will come alive and soon catch up with what has been your special pride and joy. The two accomplished ladies will be worthy of your continued confidence.

I was somewhat concerned by Dr. Morse's dim view of registries in general. It is true that those of us at the Institute have seen several registries start with enthusiasm and peter out. In fact, the Institute has salvaged several such registries. I think particularly of the Chest Tumor Registry that is affiliated with your field; the General Tumor Registry that Dr. McFarland started in Philadelphia; the Dermatologic Registry that was not too well established, and even the Bone Tumor Registry, the granddaddy of all registries in this country, has reached a state of desuetude. I am sure that Dr. Codman would rest easily if he could know of the splendid work of Lent Johnson with the orthopedic material at the Institute.

I know that General DeCoursey

would be very pleased if you would stop at the Institute and see the enormous activity that is going on there in various special fields. We are convinced that registries, properly sponsored and properly administered, can be very potent factors in education, in research, and in the advancement of the various clinical specialties.

As a result of the experience and years of contact with the various special fields, it has been evident to us general pathologists that much of the pathology of several of the specialties has been developed by clinical specialists frequently not too well grounded in general pathology. On the other hand, general pathologists have very little notion of the eye pathology and not too much of skin, bone, teeth, and so on. Well, it is our idea that with these very specialized activities at the Institute we have helped the specialist in the pathology of his field, but at the same time have importantly stimulated the general pathologist to an interest in these special fields. So that the general pathologist can talk to the eye man and to the ear, nose and throat man and the skin man in their respective lingos, that to my mind has been one of the most important results of the registries.

Medicine owes a great debt to this Academy for being the pioneer in this activity. We should honor Dr. Gradle and General Callender for fostering the idea during its early stages. It has been a great privilege of the Institute and of the Army to have participated in it.

BRITTAIN F. PAYNE, M.D., New York, N. Y.: The atlases of pathology were developed to fill a definite need for teaching material for the joint use of the American Academy of Ophthalmology and Otolaryngology and the Armed Forces of the United States.

For many years, these volumes served adequately and well, but by 1940 they were out of date. The necessity for revision was recognized by all concerned.

A committee for the revision of the atlases was appointed in 1944. It was composed of members from the Institute of Pathology and the Academy, with subdivisions for Ophthalmology and Otolaryngology.

Each member of the committee was selected to prepare and edit a section of the new atlas. By following this scheme, the work would have been completed within two years. Due to administrative changes, physical and emotional difficulties, plans were altered on several occasions.

Finally, it was decided to incorporate Dr. Friedenwald's book in the Ophthalmic Atlas under his editorship. This has proved a happy solution and increased the teaching value of the book.

The editorial committee for the Atlas of Otolaryngology, headed by Dr. E. P. Fowler, Jr., decided to continue with Colonel Ash's original work with the assistance of Dr. Raum. This work will be a great contribution in this sadly neglected field of study.

For many years, students and teachers have requested atlases to facilitate the study of pathology in Ophthalmology and Otolaryngology. As a result of this need, several works have appeared but none in the form of an atlas designed to help the teacher and student.

It has been observed that scientific works or books are almost out of date by the time they are published and distributed. Therefore, the Academy and the Institute should formulate plans for new Atlases within the next five years. Judging by the amount of time, effort and expense devoted to the present revision it is not too soon to start on the next issue.

From my experience with the present effort, I would recommend that only a single editor for each division be select-

ed to revise the revisions. The Academy should provide the technical and financial assistance necessary to make the work acceptable. The objectives of the pathology committees should include information on changes in pathology induced by such new agents as atomic energy and hormonal therapy.

It is a happy occasion for me, as former chairman of the combined committee, to know that some of the effort expended is being rewarded by the early appearance of these long-needed educational aids.

HELENOR CAMPBELL WILDER, Washington, D. C.: The illustrations for the new Atlas and Textbook of Ophthalmic Pathology might be regarded as an outgrowth of the collection of pathologic material for the American Registry of Ophthalmic Pathology. The Registry was established under the auspices of the Academy in 1922 and now includes over 35,000 accessions, accumulated largely by individual contribution of enucleated eyes and biopsies from the associated structures, but in part by the generous donation of material from the great eye centers of the country. Although these specimens were, for the most part, whole eyes or related tissues, sections of which have been prepared in the Armed Forces Institute of Pathology Laboratory, some are beautiful and unusual slides prepared in other laboratories and presented to the collection by various good friends of the Registry who believe in the importance of ophthalmic pathology.

As General DeCoursey has pointed out, there has been a constant effort to use this material, rather than allowing it to lie fallow in the files. From it the slide study sets have been made. These have been borrowed by ophthalmologists all over the country, particularly just before board examinations. Exhibits have been prepared for countless meetings and papers written on the basis of Registry material, not only by members of the staff but also by qualified investigators to whom the material is available.

Photomicrography has proved to be a most effective medium for dissemination of material from the Registry. This has been demonstrated by the DeCoursey and Ash Atlas with which you are all familiar, and on an even larger scale by the new Atlas and Textbook. With few exceptions the 955 illustrations in this volume are photomicrographs of specimens contributed to the Registry over a period of 30 years. The exceptions are the illustrations of Dr. Friedenwald's flat preparations of the retina, of Dr. Cogan's radiation cataracts, of Dr. Theobald's congenital cyst of the optic nerve with encephalocele, and the Owens' pictures of retrolental fibroplasia.

Aside from these contributed illustrations the photomicrographs have, for the most part, been selected from those taken for specific publications, such as Friedenwald's Pathology of the Eye, the Atlas of Ophthalmic Pathology by DeCoursey and Ash, and a number of exhibits and papers relating to pathology of the eye and based on Registry material, and from photomicrographs taken specifically for this new Atlas and Textbook. The last group comprises about two thirds of the photomicrographs in the new Atlas. The selection of photographs already in the files, of cases for photography from among the 35,000 in the Registry, of the best slide from each chosen case, of the best field from each chosen slide, and of the best print from each negative occupied a good many hours, not only of the authors' time but also of that of the clerical, laboratory and photographic staffs —and, of course, better cases invariably came in just as the plates were completed. They will still be there for the revisers.

There were, as you can imagine, many more discards than selections and this was not always because some photomicrographs were inferior, but because they depicted closely allied lesions. As a result there are far more pictures in the files than are represented in the Atlas.

While the Atlas has been the chief objective, the needs of those of you who conduct courses in ophthalmic pathology and those of you who attend them have not been overlooked. Color duplicates have been made of nearly every photomicrograph taken in the last 5 years—that is, the period during which work on the new Atlas has been in progress. Now there exists at the Armed Forces Institute of Pathology, a large collection of teaching material in the form of lantern slides, mostly in color. In order that these may be utilized, representative kodachromes are being copied on 35 mm. slides, and it is hoped that within less than a year loan sets will be available to those of you who are lecturing on ophthalmic histology and pathology. It is estimated that the sets will contain about two hundred 35 mm. slides each. There are, of course, many photomicrographs in the files that will not be included in the 200 lantern slide loan sets, so if any of you instructors have weak spots in your own teaching collections, it may be possible to strengthen them from the Registry collection. The slides will be of the greatest value when used in conjunction with the Atlas as many of them are described in detail therein. This new Atlas and Textbook, together with its biproduct, the lantern slide teaching sets, constitutes the most recent tangible result of your individual contributions of specimens to the Registry, and of your collaboration in its support through the sponsorship of the Academy.

MURIEL RAUM, M.D., Washington, D. C.: The selection of materials for the Atlas of Otolaryngic Pathology has presented a fascinating and challenging task — fascinating, because time and again cases of unusual interest were unexpectedly encountered—challenging not because there was a paucity of material which had to be ferreted out, but because there proved to be a wealth of material not hitherto systematically explored.

Colonel Ash, the senior author, had already written the text and done considerable work on the organization of the Atlas of Otolaryngic Pathology before recommending in December 1949 that the Academy appoint me to assist in completion of the project.

I am here this morning to discuss briefly the sources of material and technics used in preparation of the illustrations for the Atlas of Otolaryngic Pathology. I cannot let the occasion pass without stating that its completion would not have been possible without the interest, cooperation, and patience of General DeCoursey, the Director of the Armed Forces Institute of Pathology.

The collection of case histories and slides in the files of the Armed Forces Institute of Pathology was our primary source of material. These cases come from Army, Navy, Air Force, Veterans Administration, Public Health Service and civilian hospitals. This means that our material stems directly from you, the otolaryngologist, or indirectly from you, through your local pathologist.

I have reviewed large series of cases and thousands of slides to find those which were typical, those which were variations of the typical, those which were unusual or rare. A number of cases of unexpected interest were discovered. Probably as many remain uncovered, since it was impossible to review all otolaryngic cases.

Moreover, significant contributions in the form of slides of cases of unusual in-

terest were sent directly to us from individual otolaryngologists and pathologists.

We are particularly indebted to the Chairman and the members of the Otolaryngic Atlas Committee who have made notable contributions. Dr. Stacy Guild, Dr. John R. Lindsay, and Dr. Edmund P. Fowler, Jr., supplied original negatives or slides to be photographed for the section on the temporal bone. Dr. Guild devoted considerable time, as well, to the organization of this section. Many of the negatives or prints sent by Dr. Herman Semenov have been used in plates covering the normal histology of the nose and paranasal sinuses and chronic rhinitis and sinusitis. Dr. Paul Holinger and Dr. Edwin Hirsch, pathologist of St. Luke's Hospital, Chicago, loaned 40 technically excellent negatives of gross specimens which will appear in plates on the larynx, lung and esophagus.

After a case was selected for inclusion in the atlas, the material for photography was evaluated. If the slides were technically poor, additional sections were taken from the paraffin block or tissue when these were available. Many slides were faded or poorly stained, and Mr. Lawrence Ambrogi of our laboratories carefully removed the cover slip and restained the tissue on these to insure effective photography. The slide was then re-examined and the appropriate fields marked. Photomicrographs were taken at very low magnification in some instances to show anatomic relationships or to define the general topography of a lesion. Higher magnifications were used for structural and cellular detail. With the aid particularly of Mr. Charles Edwards of the photographic department several thousand photomicrographs were taken. The best were selected for use in the Atlas. Clinical pictures and gross photographs were included where available and of good quality.

The Medical Illustration Service of the Institute has been most cooperative. Many of the negatives from other sources required precise reduction for accommodation to a designated size. Streaked or discolored backgrounds on several hundred negatives had to be carefully opaqued. Complicated technics were employed to improve the quality of faulty negatives. Two plates in color are included in the Atlas: one depicting important cellular elements; the other, demonstrating the reactions of tissues to various special staining technics. Structures in many photographs, particularly those of temporal bone sections, required labels. These were painstakingly copied by an artist on the best print selected for the final plate. Artists also touched up or minimized the defects in many photographs of important cases in which a poor slide could not be duplicated.

Approximately 2,000 illustrations are included in the Atlas. To avoid monotony we have attempted to vary the arrangement of plates, and captions will appear directly under the photograph described in almost all instances.

While preparing the new Atlas, appropriate cases have been selected for a new 100 slide study set which will be available for loan. No definite date for distribution can be stated at this time since its accomplishment depends on the schedule of an already overburdened laboratory.

The Atlas has been designed for both the Otolaryngologist and the Pathologist. We hope that the Pathologist will thereby acquire a more profound appreciation of your problems, and that you, in turn will be better able to interpret his diagnosis and gain a deeper insight into his problems.

JONAS S. FRIEDENWALD, M.D., Baltimore, Md.: Dr. Roper has asked me to tell why we devoted so much of our efforts to the field of pathogenic mechanism. That puts me somewhat in the position of a graduate student defending his thesis. The easy answer would be that the chief developments in the field of pathology in the last 20 years have had to do with pathogenesis, with the mechanisms of disease processes, not merely their morphology.

More concretely, the authors faced with this assignment had a number of possible choices. We could have written an elementary textbook of morphologic pathology of the eye which the candidate for Board examinations could bone up on. Such a book might help the candidate pass the examination but it would not do much toward making him a better ophthalmologist. It would not help him think deeply about the signs and symptoms he observed in his patients in terms of the disease processes that are going on in the ocular tissues.

A second alternative was to write a reference handbook. This is a well-trodden path and though laborious would have been feasible. There is a need for such a handbook, but there are less than one hundred ophthalmic pathologists in the world who need it.

Rejecting these two conventional models we have chosen instead to attempt to present the best summary we could of current knowledge regarding the nature of the disease processes in the eye regardless of whether that knowledge was based on morphologic, on clinical, or on experimental studies. Whether we have succeeded in this effort is for you, not for us, to say.

The job was both difficult and hazardous — difficult because there was relatively little precedent for us to go on. If we had known at the outset how difficult it could be I am not sure we would have attempted it. It was hazardous, because many of the most important questions in this field lie at the very fringe of current knowledge where judgments are necessarily uncertain. I shall be much surprised if the reader of this text twenty years from now does not find much in it that will be quaintly illustrative of the ignorance of our times. In spite of these difficulties and hazards we hope that the reader will find in the book both firm knowledge and thought-provoking questions as to what is actually going on in the eyes that are under his care.

May I have one minute to say a word about my collaborators. The different authors have contributed, some more, some less, to the text, but whether their assignments were large or small they have each put their best effort into the job. They have also been extraordinarily good-natured over the hackings and shiftings which it became my responsibility to perform in the effort to convert a collection of manuscripts into a coherent book. Mrs. Wilder's contribution of the illustrations achieved an excellence which is plain for all to see. I could not possibly have gotten the manuscript ready for the press without the skilled and devoted help of Mrs. Steward. The publishers have, I think, turned out a handsome volume.

All of us who have had some share in this effort are happy to have our offspring christened before such an illustrious audience. We hope that you godparents will not disown the child when the critics have given their judgments.

EDMUND P. FOWLER, JR., M.D., New York, N. Y.: I wondered at first why I had been assigned this subject, "Histopathology in the General Medical Curriculum." I thought that everybody knew that histopathology is as important to medicine as the multiplication table is to mathematics. When I began to inquire I found out, much to my surprise, that there are real dissenters to be

found among psychiatrists, physiologists, internists, and I am sorry to say, otolaryngologists. Too often one hears "the examining boards are over-emphasizing histopathology," "insistence on morphology is stultifying medicine," "the tenets of Virchow are old-fashioned," "one must have a more dynamic approach to medicine."

not seem to realize the amount that histopathology contributes to form the background even for our newer thinking. The recent advances brought about by the isolation and clinical utilization of ACTH and cortisone would not have real meaning, except in relation to the histopathologic changes which are produced. I need only to refer to the re-

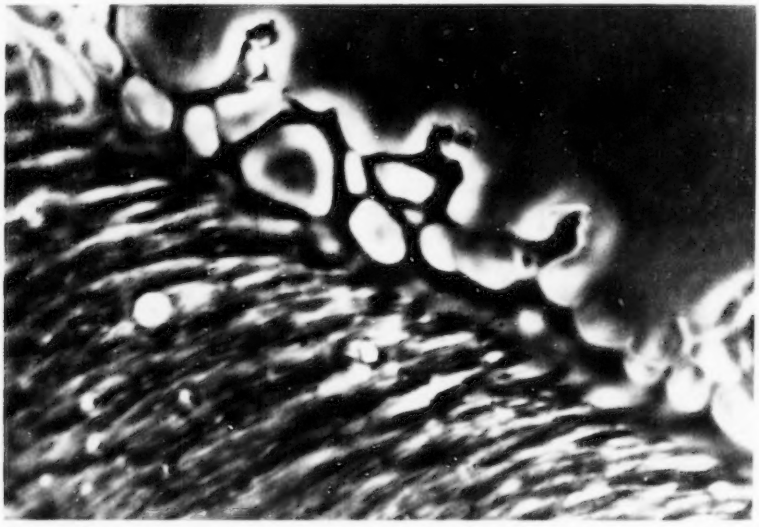


FIG. 1.—Phase microscopic picture of mobile outer edge of human tectorial membrane after it has been pulled free. Courtesy of Anderson Hilding, M.D.

However, it is still to my mind self-evident that medicine, not to mention the various surgical specialties, cannot get along without the findings of microscopic anatomy. Not only does the biopsy serve as a guide for operative procedures, but it serves also as a guide to our prognosis and therapy. It is obvious, too, that the independent diagnosis of the pathologist is essential to the maintenance of standards in all fields of surgery, as well as our own — a fact that has been recognized by the American College of Surgeons in requiring that microscopic diagnosis be made with few exceptions on all types of tissues removed at operations.

In the fields of research, too, many do

markable effects upon wound healing and upon the changes which occur in granulation tissue, changes which are related in a very intimate manner from the symptoms of patients with arthritis, not to mention allergies of the eye and nose. It may well be that the endocrinologist aided in a technical manner by the chemist, is the one who brought these discoveries to our attention. But our understanding of these discoveries would not have been possible were it not that the histopathologist had been a partner all along, and had kept pace with the work in other fields.

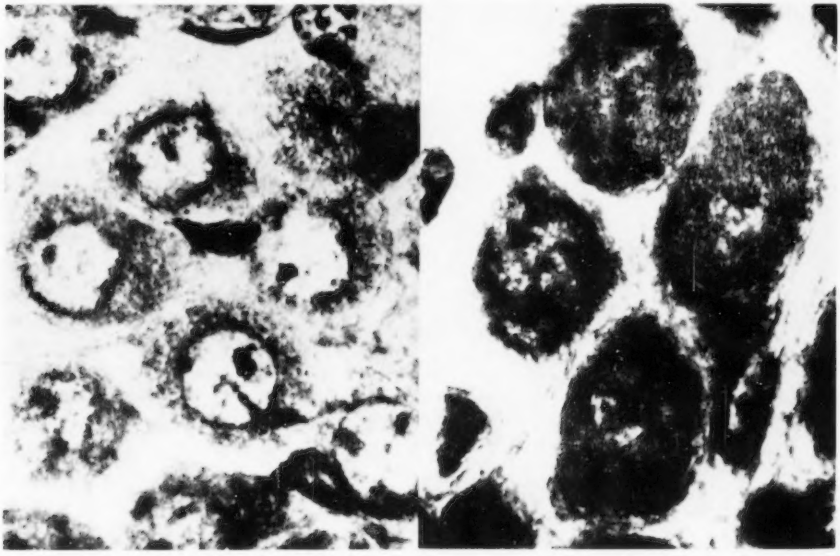
There are those who say that histopathology is dead; that there is no advance. With this I disagree. The micro-

chemical cytological ultraviolet technics of Casperson and his colleagues, as well as the new observations being made with the phase and the electron microscope show that there is no stultification in histopathology. It is hoped that further advances can be made in the study of pathologic living tissue in situ, as had been done in the edge of the spleen and in the conjunctiva and retina of the eye. Histopathology grows with the advances in other fields just as do other branches of medicine (figs. 1, 2 and 3).

I believe that it is because so few pathologic specimens of the ear and nose exist in the world that we otologists and rhinologists are comparatively backward in the understanding of our specialty. No wonder that we know so little about the common cold. We have never had a systematic histopathologic study of even this ubiquitous disease. Imagine if the eye, the kidney, or the endocrines had been so neglected. Actually, it is only when careful histopathology is done that we understand any disease.

Remember, the general pathologist cannot carry the burden of all branches of medicine alone, he does not know the special anatomy and clinical course of the case, as does the patient's own physician. So the practitioner himself must help. To do so, he must know what to look for in the office, in the operating room and in the slides. Unfortunately, there are very few pathologists who understand the intricacies of the pathology of the head and neck. We otolaryngologists should provide the specialists in this field, many portions of which are unexplored, from our own ranks.

It is for this reason that I am delighted to see the Academy reaffirm its interest in this subject by the publication of these two Atlases. I am not well qualified to speak on the eye Atlas, but it seems to me as magnificent as the ear, nose and throat one, if not as unique, and as



A B

FIG. 2.—Ultraviolet photomicrograph (4,000): (A) group of cochlear ganglion cells after being exposed to 6000 cycles at 80 db. for three hours; guinea pigs killed two weeks later. (B) Control group of cochlear ganglion cells. (From Hamberger, Carl-Axel, and Hydén, Holger: Cytochemical changes in the cochlear ganglion caused by acoustic stimulation and trauma, Acta oto-laryng., Suppl. XLI:1-89, 1945.)

FIG. 3.—Cilia from trachea of a dog (x 23,000). From Engstrom, Hans: The structure of tracheal cilia, Acta oto-laryng., 39:364-366, 1951.

useful to the general pathologist. I venture to predict that the Atlas compiled by Dr. Ash and Dr. Raum will be part of the personal library of every good general pathologist in the world, and that from its pictures will come the lantern slides, which will edify the medical students and head and neck surgeons for years to come. It should also influence the medications and judgments of those who try to avoid surgery.

I promise you that if you study, not only these Atlases, but also whatever microscopic material you can obtain from your patients, you will be richly rewarded. And remember, if you or your own pathologist cannot take care of your difficult problems, the Armed Forces Institute of Pathology will be glad to help They believe as do I, that there is no more important subject than histopathology in the curriculum of either the undergraduate or the physician at any point in his career. No practicing physician can stay up to date without it. Histopathology provides our permanent record for the basic understanding of disease.

Wherry Memorial Lecture

SOME EXPERIMENTAL OBSERVATIONS BEARING ON MÉNIÈRE'S DISEASE

JOHN R. LINDSAY, M.D.
CHICAGO, ILL.

THE subject for this lecture has been selected in accordance with a suggestion made by your program committee that it should consist of the presentation of research work carried out on a problem in the field of otolaryngology.

The experiments to be described have been done partly in the laboratory of the Department of Otolaryngology of the University of Chicago and partly in cooperation with the laboratory of physiological psychology under Dr. William D. Neff. They illustrate some of the advantages in having experimental work closely related to clinical problems, as well as examples of ways in which such problems may be attacked both by clinical and nonclinical departments.

Much of the basic research work in the field of hearing and equilibrium at the present time requires special equipment, technics and personnel and hence is usually concentrated in a few laboratories. It is our belief that such laboratories can profit from an association with a clinical department where clinical and pathological observations are in progress. In presenting the different phases of investigative work having a bearing on Ménière's disease, it should be apparent that it is the clinical and pathological investigation which indicates the existing deficiencies in our available information and points out the objectives for experimental investigation.

While a laboratory equipped for sound production, electrical recording and other special technics is essential for much of the work which may have to be done, there are many opportunities for clinical investigation and for experimental work which may not require an elaborate set-up.

Of infinitely greater value than any amount of physical equipment for the investigation of any problem are, first, a thorough knowledge of the existing factual information and, second, a fertile mind with sufficient ingenuity and drive to devise and follow up a logical method of attack. Of these two factors the second is the most important.

Clinical observations, first reported by Ménière, have long since indicated the existence of a clinical condition or syndrome with such clear-cut characteristics as to suggest a disease entity involving the labyrinth. Those features of the disease which were unexplained included the following:

a. The histopathologic picture
b. The pathogenesis
c. The etiology
d. The cause of dizzy spells, and the explanation for their great variation

From the Division of Otolaryngology of the University of Chicago.

Presented at the Fifty-Seventh Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 12-17, 1952, Chicago, Ill.

e. The impairment of hearing for low tones in early stages
f. The variations in the threshold of hearing for low tones
g. The more recently recognized characteristics of recruitment, diplacusis, decreased discrimination for speech, and the disagreeable sensation caused by sounds of high intensity

PATHOLOGY

The pathology of Ménière's disease remained a matter of speculation until the year 1938 when Hallpike and Cairns[3] first obtained the temporal bones of two known cases of Ménière's disease in which an intracranial operation had proved fatal. They found that the affected ears showed a hydrops or dropsy of the membranous labyrinth. Inasmuch as the hydrops was not associated with any other disease of the temporal bone or labyrinthine spaces, the term "idiopathic" has seemed to be appropriate for purposes of distinction from hydrops associated with other diseases.

The discovery by Hallpike and Cairns caused great interest, and in the years following there has been reported similar histopathology in over twenty ears, the majority of which have been obtained from known cases of Ménière's disease and a smaller number from cases of impaired hearing without a clear history of vertigo.

A small number of ears from cases known to have had vertigo during life have also been obtained for postmortem examination and have not demonstrated a hydrops. A scrutiny of the clinical course and findings in some of these cases suggests that they may fail to meet the rigid requirements for a diagnosis of Ménière's disease, although vertigo may have been a prominent symptom. The weight of factual information indicates that a hydrops of the idiopathic type constitutes a characteristic pathologic finding.

The inner ears which have been examined in our laboratory include three from known cases of clinical Ménière's disease and three from cases of deafness in which the history did not contain clear information regarding vertigo but included attacks of nausea and vomiting suggesting vertigo. These have all shown the characteristic hydrops of the labyrinth of the idiopathic type.

PATHOGENESIS

The pathogenesis of hydrops found in Ménière's disease has been a matter for speculation. The histologic picture has not provided the answer.

The following experiments which have been carried out in our laboratory have provided information regarding the pathogenesis of hydrops.

Experiment A

A localized injury to the bony wall of the cochlea producing a fracture or the creation of a small hole with injury of intracochlear structures and gross hemorrhage into the perilymphatic spaces has sometimes produced a hydrops. Although definite evidence of infection has not been present in these ears a low-grade infection could not be excluded as a factor.

By this means the characteristic pathologic changes produced have been fibrosis in the perilymphatic spaces, hydrops affecting the endolymphatic spaces, and extensive atrophy or degeneration of the cells of the spiral ganglion, the peripheral nerve fibers and Corti's organ.

These changes resembled those which have been well known in mild serous labyrinthitis, and also the type which have been reported in Paget's disease of the temporal bone. They present a definite contrast to the hydrops found in Ménière's disease. In the cases of Ménière's disease which I have report-

ed[7,8,10] the ganglion cells and nerve fibers have shown no evidence of degeneration due to the disease and the perilymphatic spaces have been free from fibrosis or evidence of inflammatory change.

Experiment B

A hydrops has been produced in a monkey by the procedure of fenestrating the horizontal semicircular canal and fitting a cartilaginous stopple into the window beneath the flap. The characteristic features in this case were an alteration in staining reaction of the perilymph, degeneration of the peripheral cochlear neurone and Corti's organ, and hydrops without evidence of fibrosis in the perilymphatic space except at the fenestration site. The hydrops was apparently secondary to an irritative or inflammatory reaction. The nerve degeneration was a predominant feature and apparently occurred in an early stage of the irritative process in the presence of relatively mild changes in the perilymph and an early or mild degree of hydrops.

Experiment C

A series of experiments was done to determine what changes might be produced in the labyrinth of the monkey by destruction of the endolymphatic sac and duct.[6]

If the sac and duct are important for removal of endolymph their destruction might cause retention of endolymph and, therefore, a hydrops.

In three monkeys the sac and medial dilated portion of the duct were destroyed. Over three months later the remainder of the labyrinth appeared normal with no suggestion of hydrops. A second series of experiments was carried out in the cat,[11] five ears being operated upon by the posterior fossa approach, under the dissecting microscope. Nine months after destruction of the endolymphatic sac and duct no histologic change had been produced in the remainder of the endolymphatic system, sense organs or neural apparatus. Nor was any demonstrable change in equilibrium produced.

In order to determine if any functional change might have been produced, notwithstanding the normal histologic appearance, a group of cats were conditioned to sound by Dr. Harold Schuknecht.[11] No significant change was produced in the hearing and again no vestibular disturbance was created by the destruction of the endolymphatic sac and distal portion of the duct.

These experiments had necessarily blocked off any venous channels accompanying the vestibular aqueduct, yet no functional change had resulted.

Experiment D

In another series of cats, conditioned to sound, Schuknecht[11] has been investigating the possible effect of blocking off the cochlear aqueduct on the histology and the function of the inner ear.

Up to the present the surgical blockage of this channel through which the periotic duct connects the subarachnoid space and the scala tympani has produced no disturbance of auditory function.

These four experiments have contributed certain basic information with respect to the pathogenesis of hydrops. The type of hydrops which has been found after serous labyrinthitis, leukemia, or in disease of the labyrinthine capsule, or which can be produced experimentally by an irritative reaction in the perilymphatic space as described above, differs from that found in Ménière's disease in certain essentials. In hydrops due to inflammatory or irritative reactions, the significant and early lesion consists of nerve and end organ degeneration; the hydrops is a minor accompaniment. In Ménière's disease the hydrops may become far advanced,

yet the nerve fibers and ganglion cells as well as the perilymphatic spaces and the bony labyrinthine capsule remain normal.

How can this be explained? Up to the present there is no positive information. On the basis of known facts about the behavior of semipermeable membranes, the most rational explanation would be that the chemical constitution of the endolymph becomes slightly altered by the addition of molecules which do not pass through the membranes enclosing the endolymph. The osmotic pressure of the endolymph therefore increases and is brought into equilibrium by the passage of water into the endolymph across the semipermeable membranes, thereby increasing the volume of endolymph.[8]

While such a theory may explain the pathogenesis of the hydrops, there remain several other essential points to be cleared up.

The first of these is the etiology.

ETIOLOGY

Ménière's disease is only one of several inner ear conditions in which the etiology is not clearly proven. Hence, it has been a favorite area for speculation and development of theories which have been used as a basis for therapeutic procedures.

A theory which has been revived in recent years has been that due to an autonomic imbalance a state of spasm has been produced in the arterioles which supply the stria vascularis and that the spasm is followed by venous and capillary congestion resulting in an alteration in permeability of the capillary walls of the stria and an alteration in composition of the endolymph.[4] The hypothesis of vascular spasm has been elaborated and expanded without adequate supportive evidence to include several other diseases of the ear which have not as yet been otherwise explained.

Experiments on the Blood Circulation of the Inner Ear

The available information regarding the circulation of the blood in the inner ear is based mainly on anatomic studies. The lack of physiologic information has prompted us to begin experiments on the effect of certain disturbances of the circulation on the function of the sense organ.

In view of the generally accepted idea of the blood circulation of the labyrinth, it appeared that interruption of circulation in certain channels might produce definite functional changes. For example, all authorities have seemed to agree that the arterial supply was by the internal auditory artery, a branch of the anterior inferior cerebellar in most, and in some cases a branch of the basilar artery. The venous supply has not met with the same agreement. The existence of a venous return by way of the internal auditory meatus has been described by Siebenmann and was not found by Nabeya.

The inferior cochlear vein has been accepted as an important venous channel and, according to Nabeya, the only channel for venous drainage of the labyrinth in the guinea pig and cat.

Experiments done by Perlman[15] on the guinea pig have shown that blockage of the inferior cochlear vein produced no vestibular disturbance and no diminution of nerve action potentials. The sensitivity of the recording method has been confirmed by demonstrating the effect of hypoxemia in reducing these responses, thus confirming the report by Wever.

In the cat the blockage of the venous channels accompanying the vestibular aqueduct produced no change in auditory function. Subsequent blockage of the inferior cochlear vein and cochlear aqueduct in the same animal by Schuknecht also produced no demonstrable loss of function.

It is evident from these observations on blocking the venous channels that the inner ear must be adequately provided with collateral venous drainage, and that if temporary venous congestion does occur, the sensory receptors are not readily affected.

ARTERIAL CIRCULATION

Experiments designed to alter the arterial circulation are less easily carried out since the arterial supply is less accessible.

Dr. William D. Neff and Nelson Kiang have been successful in blocking off the anterior inferior cerebellar artery, of which the internal auditory artery is a branch, in cats and in keeping the animals alive long enough to permit behavioral tests and to allow nerve degeneration to occur.

These experiments are still in progress, but the inner ear of one animal has been sectioned and shows an unusual type of lesion. In the apical and middle coils of the cochlea there were severe degenerative changes in both the organ of Corti and spiral ganglion. The hearing in these animals is being tested before and after the operative procedure. Interpretations obviously cannot be made until other ears have been examined histologically and all results evaluated. It appears, however, that the arterial supply in the cat is subject to considerable variation and that a collateral supply may exist which is adequate in many cases to maintain function when the main vessel to the internal auditory meatus has been blocked.

The effect of the autonomic nervous supply on the vessels of the stria vascularis and the sensory receptors has not yet been adequately studied. Under direct vision with the microscope the blood circulating in the smaller vessels in the middle ear mucosa can be seen to come to a standstill in some areas under sympathetic stimulation, and in some vessels the flow may reverse its direction. Upon release of the stimulus, however, the flow is resumed without evidence of injury or interference.

The effect of prolonged sympathetic stimulation on the nerve action potentials has been investigated also. Our findings have not been in agreement with a recent report by Seymour and Tappin.[19] We have found that 20 minutes of stimulation of the sympathetic supply did not decrease the amplitude of the nerve action potentials, hence probably did not cause any impairment of function. The effect of such stimulation is clearly evident as a constriction of the middle ear vessels and by dilatation of the pupil. Whether there is a similar effect upon the vessels which supply the intralabyrinthine structures is not yet known.

The results up to the present seem to indicate that the inner ear is more adequately supplied by collateral arterial and venous circulation than anatomic studies have hitherto indicated.

CAUSE OF DIZZY SPELLS

The reason for the occurrence of dizzy spells in Ménière's disease and for the unpredictable characteristics of these attacks has been of both clinical and academic interest. Two suggested explanations have been a direct effect of pressure variation on the sensory cells of the cristas and maculas and a vascular spasm of the vessels to these sense organs. Valid objections exist to both theories. As has been pointed out in former descriptions of the histopathology of Ménière's disease,[8,10] there are characteristic findings which suggest a simple mechanical explanation for dizzy attacks. These consist of distortions in the walls of the utricle occurring at the places of entry of the semicircular canals, where the walls of the utricle are thin and unsupported. Such a distortion at the ampulla of any

canal probably develops intermittently rather than gradually and must of necessity disturb the function of the ampullary mechanism since the contour of the ampulla is altered and the sliding action of the cupula probably impeded.

This simple mechanical explanation for an imbalance of vestibular function between the affected canal and the canal in the same plane on the opposite side can clarify the many unpredictable peculiarities of the dizzy spells as to the time, manner of onset, severity and duration as well as the direction of the accompanying nystagmus.

INVOLVEMENT OF LOW TONES PRIMARILY IN MÉNIÈRE'S DISEASE

The earlier theories which attempted to explain the low tone involvement in Ménière's disease on the basis of a pure inner ear conduction impairment have seemed to be ruled out with the realization that recruitment of loudness commonly present in Ménière's disease is probably characteristic of hair cell injury and does not occur in a conduction impairment.

The cause for the selective depression of low frequencies in the early phase of the disease has not yet been explained.

Localized hearing impairment for low tones due to a perceptive lesion has been produced experimentally by Schuknecht in two ways. Using cats conditioned to sound he has succeeded in producing a lesion of Corti's organ restricted to the apical coil.[16] In certain animals a hearing loss of over 50 db. limited to the low tones has been produced.

Another method by which Schuknecht has produced a localized low tone loss has been by an injury to that part of the auditory nerve leading to the apical coil.[18]

Whereas these experiments illustrate two ways in which a low tone perceptive hearing loss can be produced, in neither instance does the pathologic anatomy parallel that seen in Ménière's disease.

Variations in Hearing Threshold for Low Tones

The variation in thresholds for low tones of as much as 35 db.[9,12] depending on the temporary stage of the disease has been a matter for speculation.

Experimental evidence having a direct bearing on this phenomenon is not available.

The clinical combination of subjective increase or decrease in auditory symptoms with measurable threshold changes in hearing suggests a temporary increase or decrease in the hydrops.

If the thesis that the hydrops is due to the appearance of nondiffusable molecular substances in the endolymph and an alteration in osmotic pressure, it seems possible that the cells responsible for initiating the nerve stimulus may also be affected by such a disturbance resulting in an impairment of function.

THE RECRUITMENT PHENOMENON AND DECREASED DISCRIMATION FOR SPEECH

Clinical observations have given indication that both in a pure nerve lesion such as an acoustic neuroma and in pure conduction deafness recruitment of loudness with increasing over threshold intensities does not occur,[2] while in Ménière's disease this phenomenon is characteristic.

In Ménière's disease the histopathologic examination has shown that the cells of the spiral ganglion and the nerve fibers do not usually show evidence of degeneration. The impairment of function in this condition has been, therefore, considered as either an alteration in the conduction of the wave in the fluid or an affection of the sensory cells. Since recruitment does not occur in conduction deafness, the evidence seems to point to the hair cells as the source of hearing impairment and the recruitment.

In human temporal bones it is common to have postmortem degeneration and variations in the fixation, hence small alterations in hair cells may not be readily differentiated from postmortem changes. In early Ménière's disease with moderate hearing loss the sense organ has the capacity to return practically to normal function, hence a decrease in the number of hair cells may not be expected and a decrease in their power to function might not be detectable as a histologic change. In late stages, however, some demonstrable decrease in number or changes in the cells might be anticipated.

In one of our unilateral cases of Ménière's disease, we have been able to map out the cochlea and have found about equal numbers of hair cells and ganglion cells on the two sides. According to hearing tests made three years before death and the histopathology, this case would not be considered as advanced. However, the absence of histologically demonstrable hair cell damage is probably not incompatible with impairment of function of these cells.

Hallpike and co-workers[1,3] have reported degenerative changes in the hair cells of the affected ear which were not present in the normal ear in unilateral Ménière's disease. A review of the cases in our laboratory has disclosed somewhat inconclusive evidence of damage to the hair cells. It would seem, therefore, that these changes may be evident only in the more advanced stages of the disease or that it will be necessary to obtain autopsy material very promptly after death if minor changes in the hair cells are to be clearly demonstrated.

In our experimental material there is ample evidence that the destruction of the nerve alone may not cause degeneration of the hair cells. This was shown by Neff[13] in sound conditioned cats by crushing or cutting the nerve in the internal auditory meatus, and by Schuknecht[18] after cutting the part of the nerve leading to the apex.

Various types of injury to the sense organ, for example the experiments on acoustic trauma[14] and severe head blows,[17] have shown that a certain degree of injury is reflected only in changes in the sensory cells and their supporting structures, while a more severe injury may cause dissolution of the whole of Corti's organ and a degeneration of the nerve as well.

From the standpoint of pathology, therefore, the possibility exists of a lesion involving only the nerve, or a lesion affecting only the sensory cells, or a combination of these two. The likelihood is strong that in Ménière's disease only the sensory cells are affected.

If recruitment is characteristic of hair cell injury, it seems understandable that in certain diseases there may be various combinations of recruitment and nonrecruitment. Examples of cases showing recruitment to be present for some tones and absent for others are not uncommon in clinical practice. These would seem to be explainable as a disease beginning in the sensory cells with secondary nerve degeneration only in some areas.

The proof of the origin of the recruitment has, however, not yet been finally established by either experimental or clinical means. Other features of Ménière's disease, such as the unpleasant quality of loud sounds in some cases, diplacusis, the relatively brief duration of the recruitment effect shown by Hood[5] and the decreased discrimination for speech with increased intensity, are additional intriguing features which have combined to make this disease a challenge to everyone interested in clinical and investigative work on the ear.

In view of the many questions which remain to be answered in connection with Ménière's disease it is not surpris-

ing that we lack effective therapeutic measures. Few significant advances in basic knowledge of disease processes or in methods for their control are likely to be discovered by accident. It is by well-planned investigative work in the laboratory that additions to our factual information may be accomplished. It is desirable that such research work be carried on and supported as far as possible either within or in close association with clinical otolaryngology. Mutual benefit will result to both clinician and scientist from the understanding and free communication which is developed in carrying on a cooperative experimental investigation. The future welfare and standing of the specialty of otolaryngology will be to a great extent dependent not only upon the excellence of clinical training but on the contributions to the field of knowledge which are made from within the specialty.

REFERENCES

1. Dix, M. R., and Hallpike, C. S.: The pathology, symptomatology and diagnosis of certain common disorders of the vestibular system, Proc. Roy. Soc. Med., 45: 341-354 (June) 1952.
2. Dix, M. R., Hallpike, C. S., and Hood, J. D.: Observations upon the loudness recruitment phenomenon, with especial reference to the differential diagnosis of disorder of the internal ear and VIIIth nerve, J. Laryng. & Otol., 62:671-686 (Nov.) 1948.
3. Hallpike, C. S., and Cairns, H.: Observations on the pathology of Ménière's syndrome, J. Laryng. & Otol., 53:625-654 (Oct.) 1938.
4. Hilger, Jerome A.: Autonomic dysfunction in otolaryngology (Wherry Memorial Lecture), Tr. Am. Acad. Ophth., 56:716-723 (Sept.-Oct.) 1951.
5. Hood, J. D.: Studies in auditory fatigue and adaptation, with a preface by C. S. Hallpike, Acta oto-laryng., Suppl. 92:1-57, 1950.
6. Lindsay, J. R.: Effect of obliteration of the endolymphatic sac and duct in the monkey, Arch. Otolaryng., 45:1-13 (Jan.) 1947.
7. ———: Labyrinthine dropsy, Laryngoscope, 56:325-341 (July) 1946.
8. ———: Labyrinthine dropsy and Ménière's disease, Arch. Otolaryng., 35:853-867 (June) 1942.
9. ———: Ménière's disease, Minnesota Med., 25:778-783 (Oct.) 1942.
10. ———: Ménière's disease; histopathologic observations, Arch. Otolaryng., 39:313-318 (April) 1944.
11. Lindsay, J. R., Schuknecht, H. F., Neff, William D., and Kimura, Robert: Obliteration of the ductus endolymphaticus and cochlear aqueduct in the cat, Tr. Am. Otol. Soc. (In press.)
12. Mygind, S. H., and Dederding, Dida: The pathogenesis of Ménière's disease and of kindred conditions in the ear and the rest of the body, Ann. Otol., Rhin. & Laryng., 47:938-946 (Dec.) 1938.
13. Neff, William D.: The effects of partial section of the auditory nerve, J. Comp. Psychol., 40:203-215 (Aug.) 1947.
14. Perlman, H. B.: Acoustic trauma, Ann. Surg., 122:1086-1091 (Dec.) 1945.
15. ———: Experimental occlusion of the inferior cochlear vein, Ann. Otol., Rhin. & Laryng., 58:33-44 (March) 1952.
16. Schuknecht, H. F., and Neff, William D.: Hearing losses after apical lesions in the cochlea, Acta oto-laryng., 42:263 (June) 1952.
17. Schuknecht, Harold F., Neff, William D., and Perlman, Henry B.: An experimental study of auditory damage following blows to the head, Ann. Otol., Rhin. & Laryng., 60:273-289 (June) 1951.
18. Schuknecht, H. F., and Woellner, Richard: Partial section of the eighth nerve. (To be published.)
19. Seymour, J. C., and Tappin, J. W.: The effect of sympathetic stimulation upon the cochlear microphonic potentials, Proc. Roy. Soc. Med., 44:755-759 (Aug.) 1951; J. Laryng. & Otol., 65:851-858, 1951.

A STUDY OF THE ANATOMY AND FUNCTION OF THE TECTORIAL MEMBRANE

A. C. Hilding, M.D., Ph.D.
DULUTH, MINN.

The three points which I wish to bring out in this discussion are:

1. A description of the manner in which the tectorial membrane is inserted upon the organ of Corti.
2. The existence of an anatomic arch not previously described as such.
3. The development of a theory of the transmission of sound vibrations from the perilymph to the hair cells.

Many have suspected that the tectorial membrane is attached to the organ of Corti in some manner[12,18,20] but this is not generally accepted, nor have such attachments been satisfactorily demonstrated. Until these anatomic relations were determined, the physiologic action of the tectorial membrane and the physiology of hearing could not be clarified.[10] It is generally accepted that the "hairs" of the hair cells make contact with the tectorial membrane[5] either continuously or intermittently and that the sound waves stimulate the sense cells here, but the exact nature of the contact and the manner of stimulation have not been made clear.

The relative inaccessibility of the membranous cochlea and the significant changes in the anatomic relations produced almost inevitably during dissection probably explain the lack of information on the nature of the contact and the manner of stimulation. Fixed histologic preparations have not been satisfactory because the contents of the cochlea are altered in some of the preparatory steps.

Work done as Visiting Research Professor in the Department of Otolaryngology, Columbia University, during the 1951-1952 academic year and supported in part by grants from the Research Council of the American Otological Society and by the Hayden-Coakley Memorial Fund.

Acknowledgement is gratefully given to Dr. Edmund P. Fowler, Jr., Chief of the Department of Otolaryngology at Columbia University, for the opportunity to spend the 1951-1952 academic year in his department, where these studies were carried out.

Presented at the Fifty-Seventh Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 12-17, 1952, Chicago, Ill.

MATERIAL AND METHODS

The material used in these studies consisted in some 130 temporal bones, about 100 of which were from man. These were obtained as soon after death as possible and, whenever feasible, dissected immediately without any fixation. The apical turn, which was studied in practically all, is the most easily accessible from the vestibular side and it is possibly somewhat less difficult to save the attachments of the tectorial membrane here.

Staining was done in situ with simple stains such as methylene blue and Mallory's connective tissue stain. In this also no fixative was used.

RESULTS AND OBSERVATIONS

From these studies it became apparent that the outer margin of the tectorial membrane is inserted upon the cells of Hensen. This insertion was not to be demonstrated in any great number of specimens examined. In fact, in only 12 was an intact or fragmentary insertion to be found where attachment actually remained. After scores of dissections, it was possible finally not only to visualize the attachment of the outer margin of the tectorial membrane to the cells of Hensen but also to engage the fibrils at the upper surface of the tectorial membrane in a microhook and to manipulate the organ of Corti up and down by means of this intact attachment (fig. 1).

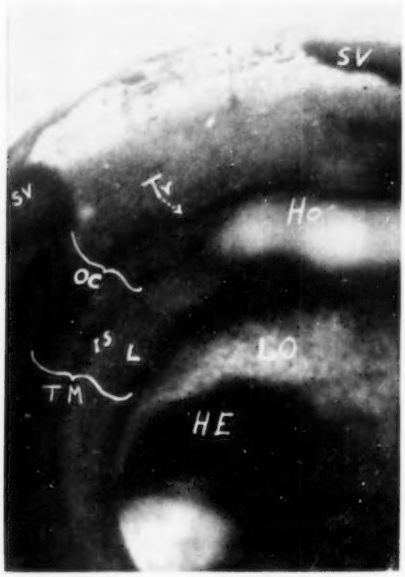


FIG. 1—Photograph showing the normal attachment of the outer margin of the tectorial membrane to the organ of Corti. A section of the stria vascularis has been removed. SV, stria vascularis; OC, organ of Corti; TM, tectorial membrane; Tr, point of traction; Ho, microhook; IS, internal sulcus; L, limbus; LO, lamina ossea; HE, helicotrema.

The outer margin of the tectorial membrane normally lies just at or just outside the outer line of hair cells. It contains a strip of homogeneous, blue-staining material (with Mallory's stain) which is continuous with what I shall, for want of a better term, call the membrane or net of insertion. The latter extends from the margin of the tectoria outward over the cells of Hensen and at least as far as the base of the latter. It seems to be similar in nature to the zone of origin on the limbus, but is much more delicate (fig. 2). It resembles a net both in situ and when torn free but may normally be a continuous membrane none the less. In situ it is deepest or thickest at the margins of the underlying cells and thinnest over the centers of these cells. When stained in situ, it shows best at the cell margins. When torn free, it is netlike in appearance but this may be due to the fact that the interstices in the net are made as it is torn free from the underlying cell bodies (fig. 3).

There is a second membrane concerned with the insertion of the tectoria which also overlies the cells of Hensen. On the inner side, this membrane extends at least as far as the outer line of hair cells and may reach farther. This second membrane is stronger than the first, just described. Under certain circumstances it can be raised from Hensen's cells, beginning at the base of the latter on the basilar membrane and tearing inward. When so raised, the netlike sheet of insertion is lifted with it and the contents of Hensen's cells balloon out as though extruded (fig. 4).

In addition to the insertion of the outer margin, Hensen's stripe seems to be similarly inserted upon the so-called border cells which lie in the internal sulcus just inside the line of inner hair cells (fig. 4). Shambaugh found this insertion in at least one laboratory animal. When the tectorial membrane is torn free, frequently numerous fragments of cells remain attached in Hensen's stripe.* These are not from hair cells, although sometimes fragments of hair cells also remain attached, but these are found in the zone between Hensen's stripe and the outer margin (fig. 2). This is the region which is normally adjacent to the hair cells and which might be called the articulating zone.

There is still another element in the insertion, a material which appears oily

* In a previous article[10] it was stated that as the tectorial membrane was torn free, the attached hair cells stretched, let go and snapped back into position. This was an error. It was the border cells which acted thus.

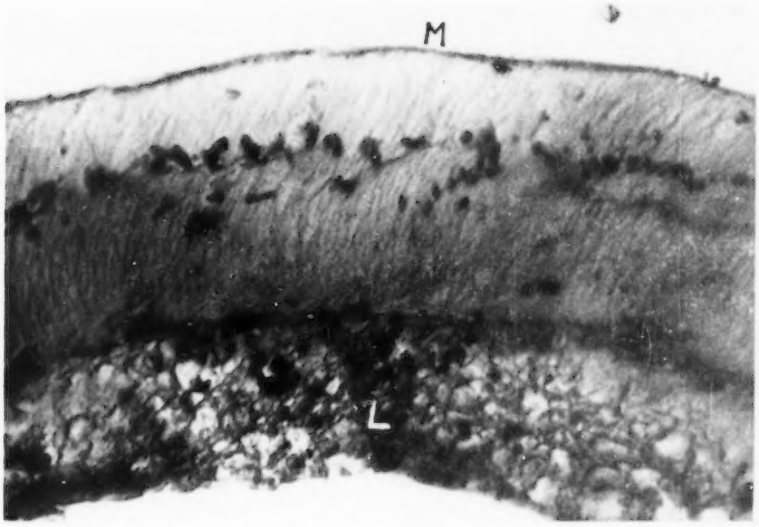


FIG. 2—View of the inferior aspect of an isolated tectorial membrane. Many cell fragments are seen still attached to Hensen's stripe. The nature of the zone of origin (L) is quite clearly shown. M, outer margin.

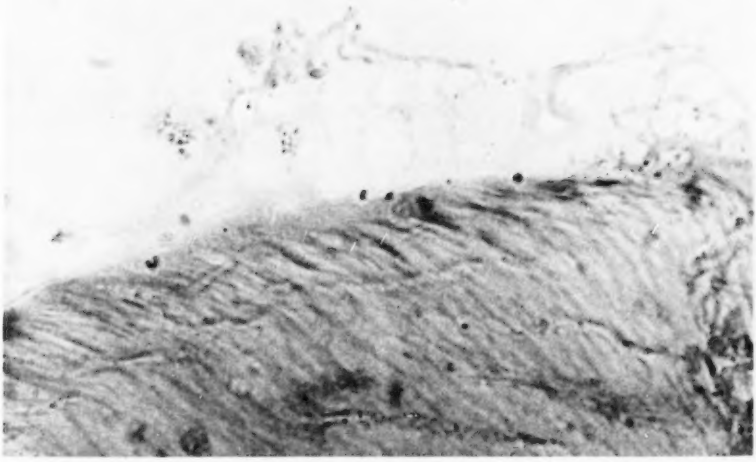


FIG. 3—View of the outer margin of the tectorial membrane with a torn remnant of the net or membrane of insertion still attached. It contains a few cell fragments.

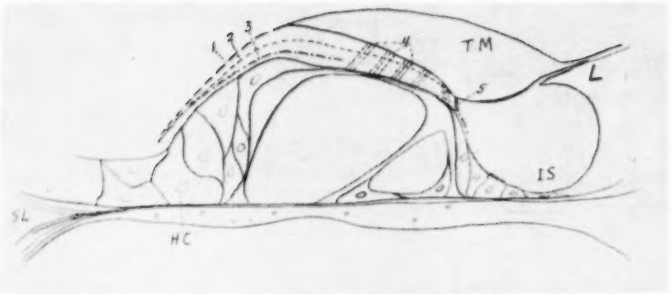


FIG. 4—Sketch illustrating the attachments of the tectorial membrane. 1. Net or membrane of insertion. 2. Material found especially between the contact zone of the tectorial membrane and the hair cells. Whether this is a lubricant or cement substance is not clear. 3. Limiting membrane overlying the cells of Hensen. When this is torn loose near the outer base of Hensen's cells, these cells tend to balloon out. 4. Attachments of the hairs of the hair cells to the tectorial membrane. The nature of this attachment is unknown. 5. Insertion of Hensen's stripe to the border cells. This insection probably contains the same elements as the one on Hensen's cells. (From Hilding[10].)

but probably is not. It occurs between the undersurface of the tectorial membrane and the organ of Corti in the form of tongues or ribbons. When the tectorial membrane is lifted, it is possible with a microhook to reach under it and engage some highly distensible fibers, which are probably identical with this material (fig. 4).

Another startling fact becomes apparent when the insertions of the tectorial membrane are studied in cross-section. There is an arch, made up of flexible parts, which fits closely over the more or less rigid arch described so well by Held.[9] It consists of three parts: the cells of Hensen, the articulating zone of the tectorial membrane and the border cells in the inner sulcus. The cells of Hensen and the border cells, which are similar in structure, are large, loose cells and in the fresh state appear to be highly mobile within a very limited range. This mobile arch fits over Held's "Stutzbogen," except that there seems to be a very narrow space between the lower surface of the tectorial membrane and the sense cells which is spanned by the hairs of the latter. This might be called the articulating space. Further, there is an arm extending from the upper member of the arch inward and attaching to the relatively flexible limbus; this is the zone of the tectorial membrane which spans the inner sulcus (figs. 5 and 6).

FIG. 5—The heretofore undescribed mobile arch outlined in heavy lines. It is composed of the Hensen's cell mass on the outside, the cell mass of the border cells on the inside and the tectorial membrane above. (From Hilding[10].)

I was fortunate in obtaining five specimens in which the movements of the basilar membrane and organ of Corti resulting from impulses in the perilymph could be observed and studied. In one, the tectorial membrane remained in situ, was well stained and could be readily observed. The outer margin was loose but, for this particular observation, the

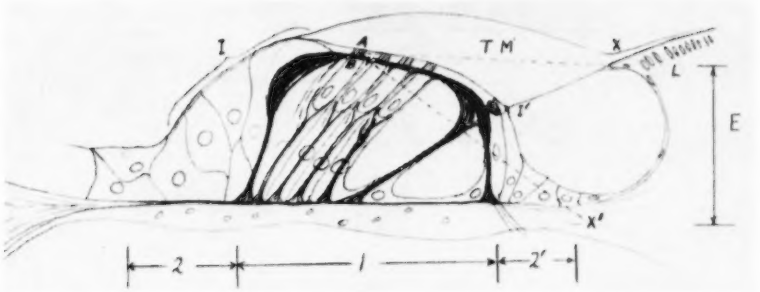


FIG. 6.—Sketch showing the relationship of the mobile arch here described and the arch of Held (Stutzbogen). The latter (1) is outlined in heavy lines, the former (2 and 2') in light lines. I and I', insertions of the tectorial membrane (TM) upon the cell masses forming the abutments of the mobile arch. E, distance between the centers of rotation of the tectorial membrane and the organ of Corti. X-X', centers of rotation. (From Hilding[10].)

fact that this margin lay free was an advantage. The basilar membrane and the organ of Corti were made to rise and fall and the action of the tectorial membrane was studied. The direction of movement of the organ of Corti was along an arc having its center somewhere near the floor of the inner sulcus. The tectorial membrane rode pick-a-back upon it without any shearing movement that could be observed. The radial movements (shearing movements) were completely absorbed by the spanning zone of the tectorial membrane, that is, the portion between Hensen's stripe and the lip of the limbus. The accordian-like action of this portion of the membrane was obviously sufficient to neutralize any radial movement of the organ of Corti, thus preventing a shearing motion between the tectorial membrane and the hair cells.

The slow, artificial movements used in this study did not produce any observable slopes directed either toward the apex or base. As far as could be determined, the entire length of the basilar membrane rose and fell. No movement in the axis of the fibrils was observed.

From these studies, the writer has concluded that the tectorial membrane is inserted upon Hensen's cells at its outer margin and upon the border cells at Hensen's stripe and that the zone between is an articulating zone where the "hair cells" are attached, or at least are in continuous contact.

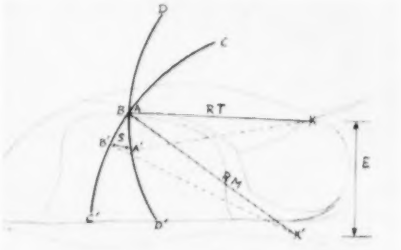


FIG. 7.—Diagram illustrating the movements of adjacent points, A on the tectoria and B on the organ of Corti, during up and down movements, if both were rigid. RT, radius of the arc D D' described by point A on the tectorial membrane as it moves about its center of rotation, K. RM, radius of the arc C C' described by point B on the organ of Corti as it moves about its center of rotation, K'. S indicates the separation of the points A and B when depressed to the position A' B'. (From Hilding[10].)

THEORY

There have been so many theories of hearing suggested during the past three hundred years that one hesitates to propose another. Recalling that many of these theories have been developed by the finest minds of their generation, the subject is approached with trepidation.

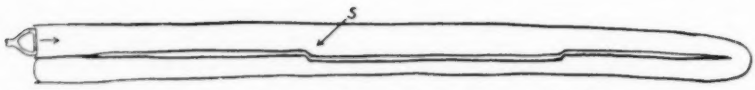


FIG. 8—Sketch of the cochlea unrolled. The central line represents the basilar membrane and the one above it represents the tectorial membrane. When an impulse coming from the stapes depresses a portion of the tectorial membrane and basilar membrane, these structures are found simultaneously in four positions: the original position, two slopes, one facing the base and one facing the apex, and a depressed zone between. The slope facing the apex (S) is the only point at which traction is made by the fibrils in the tectorial membrane.

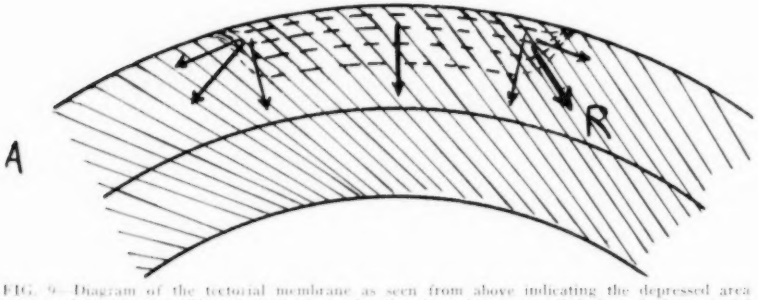


FIG. 9—Diagram of the tectorial membrane as seen from above indicating the depressed area illustrated in figure 8. The arrows indicate the direction of forces applied through the tectorial membrane. All of these forces cross the axes of the fibrils except the one at R. This is the only direction in which a pull through the fibrils would be exerted. (From Hilding[9].)

Yet, with these new facts in hand, one cannot avoid pondering their significance, especially in view of the many other established facts which do not find a place in most theories of hearing.

Let us take the fact that the origin of the tectorial membrane lies at a higher level than the center of rotation of the basilar membrane as the point of departure. In up-and-down movement, the organ of Corti and the tectorial membrane, riding upon the basilar membrane, would each describe an arc. These two arcs would not coincide because the center of rotation of one (the tectorial membrane) lies at a higher point than that of the other (the basilar membrane). Hence, if all of the parts involved were rigid, a shearing motion between them would inevitably result (fig. 7). When actually observed, there was no such shearing movement when the parts moved up and down. The spanning portion of the tectorial membrane was sufficiently yielding to push and pull to absorb the movement.

Let us now make a hypothetical picture and assume an impulse comes into the scala vestibuli from the oval window, depressing a relatively large part of the tectorial membrane and the basilar membrane. Then the tectorial membrane and underlying organ of Corti would assume four different positions simultaneously: (1) the unaffected original position of rest, (2) a slope downward facing the apex, (3) a slope downward facing the base and (4) a flat depressed region between the two slopes (fig. 8). The depressed region which lies in a lower plane is flat because the action of the upper arm of the spiral ligament checks the downward movement.

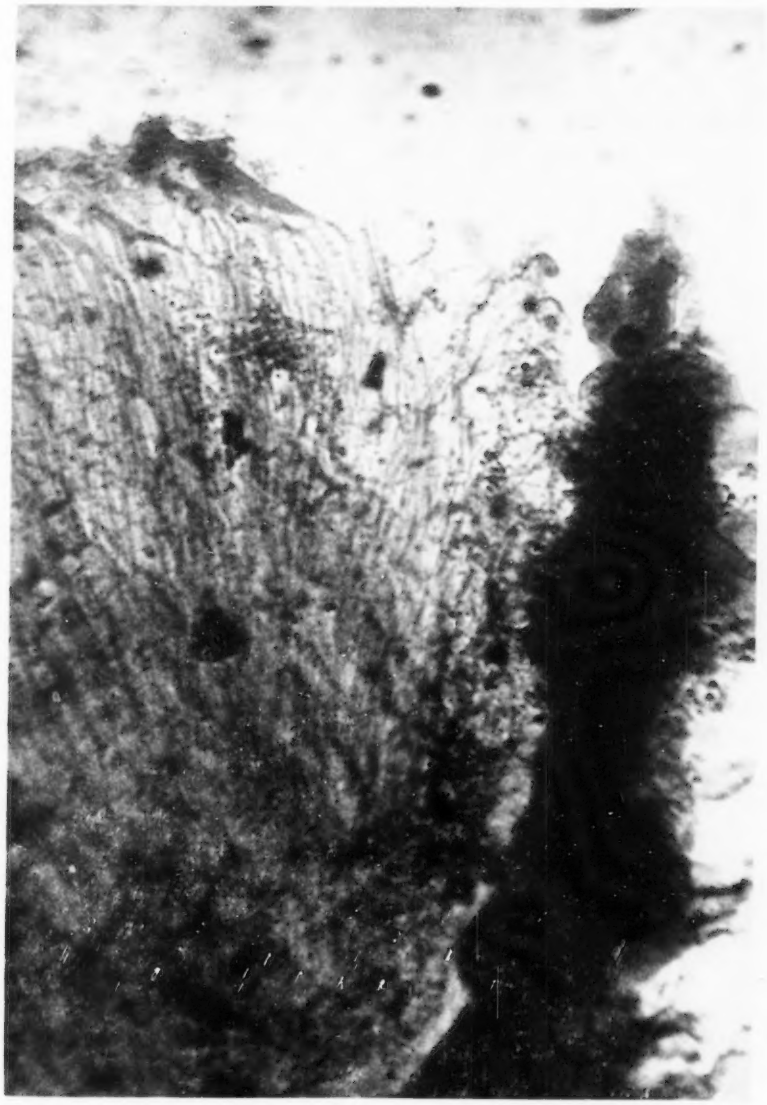


FIG. 10—Photograph of the tip of the tectorial membrane showing some of the fibrils teased out. The exact nature of these fibrils is not known. They may be tubes and they may be spiral in form, similar to those found in the cupula.[6] The dark mass at one side is the termination of the zone of origin.

The shearing force through the entire length of the depressed region would result from a pull by the tectorial membrane and would be directed radially toward the modiolus. On the slope facing the base, there would be a force directed toward the modiolus, and because of the thickness of the tectorial membrane, there would be a second axial force directed toward the apex. The resultant of these two forces, if they were equal, would be directed about 45 degrees inward and toward the apex (fig. 9). Similarly, on the slope facing the apex, there would be two forces acting, the resultant of which would be directed inward and toward the base. The exact angle would depend upon the relative magnitude of the two forces and the amplitude within which they were effective.

If the tectorial membrane is examined, it is found to contain fibrils* which are almost parallel (fig. 10). They are directed toward the apex and outward at an angle of about 30 or 40 degrees. These fibrils appear to be nonstretchable, as evidenced by the following facts: (1) when grasped and pulled longitudinally, the tectorial membrane does not stretch, but it is comparatively nonyielding; (2) the membrane yields more readily to a transverse pull, across the fibrils; (3) point pressure on the upper surface makes an oval dimple, the long axis of which parallels the fibrils; and (4) when a radial pull directed toward the modiolus is exerted upon the upper surface of the tectorial membrane, the retraction at the margin extends much farther toward the apex than toward the base. In our hypothetical picture, the resultant shearing force on the slope which is directed toward the apex would lie about in the axis of these fibrils, while in the other three positions the shearing forces would be directed across the fibrils at angles varying from 60 to 90 degrees (fig. 9). Herein lies the central point of the theory which I am postulating. If the fibrils are nonyielding and do not stretch, then on this slope facing the apex, and nowhere else, a shearing movement would result from the shearing force, because the fibrils are relatively fixed at their origin. Such a movement would cause a pull upon the insertion of the fibrils and upon the underlying attached hairs of the hair cells. This, in my opinion, is the localized stimulating movement for which we have been looking. If the tectorial membrane is sufficiently yielding transversely across the fibrils to absorb the shearing forces directed across the axis of the fibrils, as it appears to be, then no pull would be exerted upon the insertion or upon the hair cells at any other point.

In our hypothetical picture, then, of a downward movement of a large portion of the tectorial membrane, organ of Corti and basilar membrane following an impulse in the scala vestibuli, the only point at which a movement would result between the tectorial membrane and the hair cells would be on the slope directed toward the apex, where the direction of the shearing force and movement coincides with the axis of the fibrils. The crux of the matter lies in the comparative flexibility of the tectorial membrane across the fibrils as compared with its flexibility in the axis of the fibrils. (Flexibility is here used to denote the quality of yielding to push and pull.)

If this concept is correct, then a stimulating movement between the tectorial membrane and the hair cells results from sound vibrations in the perilymph of the scala vestibuli about as follows: A wide sector of the basilar membrane, organ of Corti and tectorial membrane

* The nature of these so-called fibrils is not clear. They might even be tubes.

is thrown into vibration and a wave movement in these structures results. This wave is characterized by a sharp break or slope directed toward the apex. The organ of Corti with the articulating zone of the tectorial membrane rises and falls upon an arc with its center near the tympanic lip of the limbus (at or near the inner attachment of the basilar membrane). Except at one point, the movement in the radii of the cochlea (closer and further away from the vestibular lip of the limbus) is absorbed by the yielding spanning zone of the tectorial membrane. The spanning zone is the portion between the vestibular lip and the stripe of Hensen. No shearing movement between the tectoria and hair cells occurs excepting in the small area where a slope toward the apex is found, and here there is a diagonal shearing movement in the axis of the curved, arching fibrils. When the basilar membrane moves down, these nonyielding, curved fibrils are pulled across a barrel, so to speak, because their origin, near the margin of the lamina ossea, is nonyielding. They, in turn, exert a pull at the point where they terminate in the tectorial membrane at or near the hairs of the hair cells. This pull is directed inward and toward the base. In addition, the pull is transmitted to the insertions of the tectoria at the stripe of Hensen to the border cells and at the outer margin to the cells of Hensen. These mobile cells yield, causing a shearing, sleeve-like movement of the mobile arch, which is directed diagonally inward and toward the base. This, in turn, is transmitted to the hairs of the hair cells, which are relatively fixed in the "Stutzbogen" of Held. They may be further braced against the pull of the fibrils by the diagonally placed phalangeal processes of the cells of Deiter. The mass of Hensen's cells is larger than the mass of the border cells because the up-and-down movement near the middle of the basilar membrane, where these cells lie, is much greater in amplitude than it is in the inner sulcus where the border cells are found. The former must accommodate to a larger amplitude than the latter. Larger shock absorbers are needed where the movement is greater.

In the upward movement, the structures move back into their original position. This is the movement of recovery and is brought about by the mobile cells in Hensen's group and the border group and probably by the criss-cross fibers in the body of the spiral ligament. The downward movement is limited in extent by the check ligament action of the upper limb of the spiral ligament and the upward movement is similarly limited by the checking action of the lower limb of the spiral ligament. (The spiral ligament seems to exert sufficient tension to hold the basilar membrane in a plane when at rest.) As the spanning portion changes in thickness during the oscillations, it is accommodated by the mobile fluid in the moat in the inner sulcus.

This description of the transmission of sound vibrations from the perilymph to the hair cells is undoubtedly oversimplified and incomplete. There must be further narrowing of the effective shearing movement on the slope directed toward the apex. This could be brought about by transverse waves and/or by further selection within the organ of Corti by means of its peculiar system of braces and tunnels.

The hypothetical picture which we created for the purpose of illustrating the principles involved probably does not exist. Instead of a depressed zone, the true picture would be that of a rising wave with a long slope directed toward the base and leading up to the crest from which a steep slope directed toward the apex would lead down again to the relatively stationary region be-

FIG. 11—Diagram illustrating the probable approximate shape of the stimulating waves in the basilar membrane, organ of Corti and tectorial membrane. The effective point of stimulus would be at S. The size of the wave as indicated is, of course, exaggerated.

yond. The stimulating movement would occur on this slope of the wave and the recovery would occur in the reverse phase when the wave is down with the steep slope directed toward the base (fig. 11). The position of the effective slope in the cochlea would vary with pitch. The higher the pitch, the closer it would approach the base.

SUMMARY

From a study of the attachments of the tectorial membrane, carried out by dissection of some 130 temporal bones —mostly fresh—it has become apparent that the tectorial membrane is inserted upon the organ of Corti. The outer margin is attached to Hensen's cells by an extremely thin membrane or net and the undersurface along Hensen's stripe appears to be attached to the border cells in the inner sulcus. The zone between these two insertions is in contact with the hairs of the hair cells. There is a material found between the undersurface of the tectorial membrane and the reticulum which seems to be fluid or semifluid in nature. This substance probably acts as a lubricant or roller bearing between these two surfaces in an articulating space.

A previously undescribed arch, which fits closely over Held's "Stutzbogen," is demonstrated. It is composed of the border cells on the inside, the articulating zone of the tectorial membrane above and the cells of Hensen on the outside. This arch is mobile within a very limited range.

A theory of the transmission of sound vibrations to the hair cells is developed. This theory includes not only the anatomic facts disclosed by the reported study but embraces a number of other known anatomic facts. The shape of the waves produced in the basilar membrane by sound vibrations, as reported in the studies of the qualified workers, also fits into this concept.[1,2,3,17,19] The transmission of sound vibrations is postulated by this theory to occur through a diagonal shearing movement resulting from a pull on the fibrils in the tectorial membrane. These originate at a higher level than the pivot point in the basilar membrane. This pull is confined to one small area where the structures involved are tipped toward the apex by the wave movement. Elsewhere, the shearing movement is neutralized by the yielding quality of the spanning zone of the tectorial membrane.

BIBLIOGRAPHY

1. Békésy, V. G.: Contribution to theory of hearing. Mode of vibration of the basilar membrane, Phys. Zeitschr., 29:793, 1928.
2. ———: The variation of phase along the basilar membrane with sinusoidal vibrations, J. Acoustic. Soc. Am., 19:452, 1947.
3. ———: Direct observation of the vibrations of the cochlear partition under a microscope, Acta oto-laryng., 42:197, 1952.
4. Bethes, A.: Handbuch der normalen und pathologischen Physiologie, 11:497, 1926.
5. De Vries, H.: Struktur und Lage der Tektorialmembran in der Schnecke, untersucht mit neueren Hilfsmitteln, Acta oto-laryng., 37:334-338, 1949.
6. Dohlman, Gösta: Personal communication to the author.

7. Hardesty, Irving: On the proportions, development and attachment of the tectorial membrane, Am. J. Anat., 18:1-73, 1915.

8. ———: On the nature of the tectorial membrane and its probable role in the anatomy of hearing, Am. J. Anat., 8:109-179, 1908.

9. Held, Hans: Handbuch der normalen und pathologischen Physiologie, 11:467-534 1926.

10. Hilding, A. C.: Studies on the otic labyrinth. I. On the origin and insertion of the tectorial membrane. II. A theory on the stimulation of the organ of Corti by sound vibrations, Ann. Otol., Rhin. & Laryng., 61:354-383, 1952.

11. Kishi, K.: Corti'schen Membran und Tonempfindungstheorie, Arch. f. d. ges. Physiol., 116:112-123, 1907.

12. Kolmer, W.: Möllendorff's Handbuch der mikroscopischen Anatomie des Menschen, Berlin, Julius Springer, 3:342-346, 1927.

13. ter Kuile, Emile: Die Uebertragung der Energie von der Grundmembran auf die Haarzellen, Arch. f. d. ges. Physiol., 79: 146-157, 1900.

14. Meyer, M. F.: How We Hear, Boston, Charles T. Bandford, 1950, p. 37.

15. Mygind, S. H.: Experimental-histological studies on the labyrinth. XI. Correlation of our histological and clinical experiments and observations, Acta oto-laryng., 35:108-129, 1947.

16. Perlman, H. B.: Observations through cochlear fenestra, Laryngoscope, 60:77-96 (Jan.) 1950.

17. Peterson, L. C., and Bogart, B. P.: A dynamical theory of the cochlea, J. Acoustic. Soc. Am., 22:369, 1950.

18. Prentiss, C. W.: On the development of the membrana tectoria with reference to its structure and attachments, Am. J. Anat., 14:425-459, 1913.

19. Ranke, O. F.: Theory of operation of the cochlea: a contribution to the hydrodynamics of the cochlea, J. Acoustic. Soc. Am., 22:772, 1950.

20. Shambaugh, G. E.: A restudy of the minute anatomy of the structures in the cochlea with conclusions bearing on the solution of the problem of tone perception, Am. J. Anat., 7:245-257, 1907.

21. Wegel, R. L.: Physical data and physiology of excitation of the auditory nerve, Ann. Otol., Rhin. & Laryng., 41:740-779, 1932.

22. Wever, E. H.: Theory of Hearing, New York, John Wiley & Sons, 1949.

23. Wittmaach, Karl: Uber Bau und Funktion der Membrana tectoria, Acta oto-laryng., 24:397-423, 1936.

24. Zwislocki, J.: Theory of the acoustical action of the cochlea, J. Acoustic. Soc. Am., 22:778, 1950.

DISCUSSION

EDMUND P. FOWLER, JR., M.D., New York, N. Y.: I have had the privilege of seeing several of Dr. Hilding's dissections in the fresh state and have watched him tear off the attachments of tectorial membrane with a microhook. I have seen him try to make the organ of Corti move with stimuli from a medicine dropper in a micromanipulator. Once I have seen it rocking on its axis as described by Dr. Hilding this afternoon.

I should like to show you my impressions of what I saw. Unfortunately, Dr. Epanchin, our medical photographer, was not free to take moving pictures of some of Dr. Hilding's crucial experiments. But we have made a model which shows shearing action on the hair cells, such as seems to occur in the parts of the cochlea which move with changes in the pressure of the perilymph.

According to Dr. Hilding's observations only comparatively small areas of Corti's mechanism move under the stimuli he has applied. Whether this is the same with sound is yet to be proved.

To my mind the important parts of this paper are the definite findings of attachments for the tectorial membrane, not only to the cells of Hensen at its outer end but also to the inner sulcus cells which spiral along with the inner hair cells.

This makes the tectorial membrane act in essentially the same manner as the cupula, or perhaps more as does the macule of the utricle, as might be expected. When the Lord finds a good mechanism He uses it over and over again, changing only secondary details to arrange for necessary changes in function. Actually it was this concept that brought Dr. Hilding to my laboratory. Both of us, separately, on seeing the experiments of Gösta Dohlman on the cupula of the codfish felt that the tectorial membrane must work in the same way and we always wanted to prove it. Dr. Hilding took the time and it was my pleasure that Columbia University was able to give him some facilities to do it.

HENRY B. PERLMAN, M.D., Chicago, Ill.: Dr. Hilding is concerned with a fundamental question. Despite a great deal of thought and experimentation, we do not know how the organ of Corti works. The complicated construction of this mammalian end organ appears to be largely concerned with a further refinement of the physical stimulus over that in the similar but simpler end organ in birds. Many of the component parts of this very complicated end organ have been assigned a theoretical role in controlling the stimulus to the hair cell. Dr. Hilding has directed his attention to the tectorial membrane and his work emphasizes the value of studying the fresh labyrinth for a better understanding of the true relations, physical properties and function of gelatinous materials like the tectorial membrane and cupular substance. Similar studies in recent years have led to a better understanding of the structure and function of both the vestibular and auditory end organ. Dr. Hilding has advanced our knowledge of the true anatomy of the tectorial membrane. The detailed observations on the origin and insertion of the tectorial membrane are new and indicate the exceedingly painstaking, delicate work that was carried out by him in this study.

In the course of some studies on the fresh cochlea during acoustic stimulation, I have obtained some general impressions about the structure, relations and function of the tectorial membrane. In suitable fresh preparations, the tectorial membrane is always attached to the underlying cells. No free edge is observed. It is a rather stiff gelatinous ribbon that adheres to the softer cells below but can be lifted away starting from its external attachment in the region of Hensen's cells. It separates in relatively long ribbons. The membrane bends most readily in the transverse direction and breaks along a transverse line at right angles to its long axis. In response to acoustic stimulation the membrane vibrates at right angles to the cochlear duct similar to the basilar membrane. The vibration of the latter pivots at the bony spiral lamina. The fluid adjacent to these vibrating membranes is set into active rotation at right angles to the cochlear duct. Strong, low frequency stimuli (i.e., 100 cycles) are observed to cause a very extensive vibration. With very strong acoustic stimuli the tectorial membrane can be thrown free of the underlying cells and its attachments. When so loosened it may stay elevated while vibrating and then slowly sink down on the supporting cells when the sound is turned off. The use of specially prepared highly reflecting silver crystals about 0.04 mm. in diameter facilitates observations of the movement of the membrane as well as of the fluids adjacent to it during sound stimulation.

In addition to direct examination, the relations of the tectorial membrane to the rest of the organ of Corti were studied in our laboratory with the help of Dr. De Vries, by freezing the cochlea in liquid nitrogen, cracking it and then thawing the fragments in Ringer's solution at room temperature. Other fragments were examined after dehydration at 30 degrees below zero. In another series of experiments carried out with the cooperation of Professor Gersh from the Department of Anatomy and Mr. Robert Kimura, frozen dried preparations of the bat cochlea were imbedded in celloidin and microscopic sections were made without decalcification. All of these studies confirm Dr. Hilding's observation that the tectorial membrane is normally in contact with the cells of the organ of Corti. In the light of our present understanding of the true relation of this membrane it might be profitable to designate the area between the tectorial membrane and the basilar membrane with its complicated series of fluid channels and cellular elements as the scala interna, so as to separate it from the rest of the scala media between the tectorial and Reissner's membrane. Any brief discussion of a theory of cochlear function is difficult in the light of the many theories that are currently defended. Dr. Hilding's theory about the function of the tectorial membrane is concerned with the ever-recurring problem of explaining our keen sense of pitch perception entirely on the basis of mechanical frequency analysis by the cochlea. He attempts to limit the effective action of the observed, broadly vibrating tectorial membrane only to the apical end by assigning a special role to the apically directed, relatively nondistensible diagonal fibers in the upper surface of the tectorial membrane. These fibers are supposed to control a diagonal shearing force by being in line only with the sloping edge of the widely moving basilar membrane. However, he illustrates clearly how the differences in the pivot for movements of the tectorial and basilar membrane would ordinarily tend to produce a transverse shearing force to the hair cell but contends that the special manner of attachment of the undersurface of the tectorial membrane to the hair cell allows the rest of the widely vibrating tectorial mem-

brane to ride with the hair cells so that no shear occurs. Direct observations of the structures within the cochlea during acoustic stimulation do not suggest these different modes of vibrations of the tectorial membrane. The increasingly wide area of vibration of the basilar membrane observed with low frequencies of increasing intensity is more in line with the increasingly broad masking effect of such stimuli rather than with restriction of the stimulation to only the apical end of this broadly vibrating structure. Furthermore, Békésy's experiments suggest that a transverse shear across the organ of Corti produces the largest electrical response.

We are indebted to Dr. Hilding for his new information about the anatomy of the tectorial membrane and for the stimulation of a theory of its function. I hope that his observations on the tectorial membrane during acoustic stimulation will further clarify this theory.

DR. HILDING: I want to thank the discussers very much for their remarks and I want to take this occasion to thank Dr. Fowler for the great privilege of working in his department for a year. He invited me to come from Duluth and spend a year in New York in his department. It was a wonderful year of study and I am most grateful.

Dr. Perlman speaks of the right-angled orientation of the different parts in the scala media. It is true that the parts are oriented at right angles to the long axis but there are two notable exceptions. The fibrils in the tectorial membrane, as I measured them in situ with the outer margin still attached, lie at an angle of about 30 degrees to the tangent of the curved axis. They extend outward from their origin and toward the apex.

There is one other exception. In the fibrous arch of Held, there are phalangeal processes which pass upward and toward the apex from Dieter's cells. Their angle is also about 30 degrees but they are directed inward as if to meet the fibrils of the tectorial membrane. They might well act as braces against the diagonal pull of the fibrils, as I have tried to explain it. In any case, the diagonal direction of these structures is a fact.

I am not quite sure that Dr. Perlman understood what I meant about the fibrils. I did not mean that only those on the surface were active. I'm afraid that might be inferred from what I said. As a matter of fact, the active ones are probably not at the surface, but in the body and deep down.

In Békésy's experiments in which transverse action gave the greatest electrical potentials one would have to know if the outer margin was attached and if he was pulling just directly across the cells. As I told you, the fibrils in the outer margin tend to retract toward the base when detached, so that they would become oriented more transversely.

Since this theory of mine is a theory only, like all theories it is, of course, suspect. I hope that all of you feel free to snipe at it. I shall join you. I am going to try not to defend it. If it is correct, it will stand on its own feet. One friend met me here in the hall yesterday and asked me if I believe it myself. Well, that depends upon what you mean by "believe." I don't "believe" that a phonograph needle can transmit the sound of an eighty-piece orchestra. That seems so impossible that it looks silly—one little point transmitting simultaneously all the different tones of eighty instruments. Yet the stubborn fact is that it does just that. Facts are terribly stubborn.

If I had been present when the Master Engineer laid out His blueprint for designing the ear, I would have told Him that it wouldn't work; it couldn't possibly work. A clumsy thing like fluid lying as a single unit on top of five thousand tiny, delicate, individual hearing units can't sound them individually. But the stubborn fact is that it does—the entire instrument works and with unbelievable sensitivity. One can only stand and marvel and exclaim like the psalmist, "Great and Glorious Are Thy Works, O God." We try in our feeble way to understand partially, and that is what I have tried to do in these studies.

TUMORS OF THE NECK

LOUIS H. CLERF, M.D.
PHILADELPHIA, PA.

TUMORS or swellings of the neck often present difficult problems in diagnosis. While many may be asymptomatic, their presence is a source of concern to the patient and his family. They may represent the first demonstrable evidence of serious disease of the upper air or food passages or they may be local manifestations of a systemic disorder. As examples may be cited the occurrence of a painless lump in the course of the upper cervical chain of lymph nodes representing metastasis from a pharyngeal carcinoma and the enlargement of several lymph nodes associated with fever and malaise as the first evidence of infectious mononucleosis. It is imperative, therefore, that a careful study of swellings of the neck be made before any plan of therapy is instituted, since the multiplicity of causes also suggests that a variety of therapeutic procedures may be indicated. As a rule, a correct diagnosis can be made by careful history, physical examination, roentgen and systemic studies and, in certain instances, biopsy.

While some tumors are of congenital origin, they may be observed at any time from birth to old age. An understanding of the embryologic development of the structures in the neck is helpful in diagnosis as well as in treatment. Among the lesions that are congenital anomalies or vestigial rests are thyroglossal tract cyst and fistula, aberrant thyroid tissue, aberrant parathyroid adenoma, aberrant salivary tissue, branchiogenic cyst, branchiogenic fistula, and dermoid cyst.

Presented at the Fifty-Seventh Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 12-17, 1952, Chicago, Ill.

FIG. 1.—A boy, 6½ years of age, who had developed a swelling of the right side of the neck three months previously, presumably complicating influenza and measles. There was some impairment of hearing. Examination of the nasopharynx revealed a tumor mass, which was biopsied. The diagnosis was lymphoepithelioma. The case was not one for surgical treatment because of the location and the extent of the disease and irradiation was carried out, but the outcome was unfavorable.

Among the acquired tumors of the neck, enlargement of lymph nodes is most often observed. They may represent a simple lymphadenitis, be part of a systemic disorder, or be of tuberculous, granulomatous or neoplastic origin. They may be single or multiple and, considering the distribution of lymphatics in the face and neck, may involve the submental, submaxillary, superficial or deep cervical chains, in fact any part of the anterior or posterior triangles of the neck (fig. 1). In addition, many benign

FIG. 2.—A child, 3½ years of age, who developed a swelling on the front of the neck. This exhibited inflammatory changes after three weeks, and a small fistulous communication which drained mucoid material developed. The patient was sent in with a diagnosis of infected thyroglossal cyst. A roentgen study revealed a widely opened, deformed safety pin in the hypopharynx and larynx. The point shaft, directed anteriorly between the arytenoids, had penetrated the anterior wall of the trachea below the level of the vocal cords. There was no history of choking on a foreign body nor was there dyspnea or hoarseness.

tumors of the skin and subcutaneous tissues including sebaceous cyst, lipoma, hemangioma, hygroma, and fibroneuroma are observed, as well as tumors of the thyroid gland.

The location of the tumor is of considerable importance in diagnosis. A swelling occurring in the midline may be a thyroglossal cyst or fistula, dermoid cyst, adenoma of the thyroid gland involving either the pyramidal lobe or the isthmus and lymphadenopathy. Invasion of the pre-epiglottic space by an epiglottic carcinoma with swelling over the thyroid cartilage and prelaryngeal abscess secondary to a laryngeal or tracheal foreign body have been observed (fig. 2).

In the submaxillary area, enlargement may be the result of obstruction to the duct of the gland by calculus. Fluctuation in size, particularly when eating, is significant. The most common neoplasm of the submaxillary gland is mixed tumor (fig. 3).

FIG. 3.—A boy, age 5 years, with swelling in right submaxillary region which had persisted for over six months. Examination indicated that it was in close relation to the posterior part of the submaxillary gland. No calculus was present. The mass was removed and was found to consist of a large multicystic tumor which occupied the posterior two thirds of the gland. The anterior third proved to be normal submaxillary gland. The histologic diagnosis was a simple cyst.

In the lateral portions of the neck there may be present sebaceous cyst, dermoid cyst, fatty tumor, hemangioma, branchial cleft cyst or fistula, cystic hygroma, carotid body tumor, aneurysm of the carotid artery and lymphadenopathy of infectious origin or due to a metastatic lesion, Hodgkin's disease, lymphoma, lymphosarcoma, lymphoepithelioma, syphilis, tuberculosis or infectious granulomas.

In the supraclavicular area there may be present a lymphadenopathy (Virchow's node), subclavian aneurysm, primary pulmonary sulcus tumor, Hodgkin's disease, lymphosarcoma, fibrolipoma, hemangioma, tumor of the thyroid gland, herniation of the lung and metastasis from laryngeal carcinoma.

Inspection aids enormously in diagnosis. Movement of the mass with swallowing usually indicates that the lesion is of thyroid gland origin, and this would include thyroglossal cyst or tumor of the thyroid gland itself. Movement of the mass when protruding the tongue suggests a thyroglossal cyst or fistula.

Palpation is an important diagnostic aid and helps to confirm the site of the tumor, its consistency, shape, size and relation to fixed points. Bimanual palpation should always be practiced in tumors involving the submental and submaxillary region.

Auscultation is of value, particularly in vascular lesions. Pulsation may be transmitted or expansile and should be differentiated. Transillumination is useful, particularly in cystic hygromas and in large thyroglossal cysts.

Roentgen study always should be made. While it is not helpful in many of the lesions, it occasionally gives valuable assistance. In the case of fistula the injection of a radiopaque substance and roentgen study is of value in arriving at a diagnosis and also in determining the extent and direction of the fistula.

While there is a great temptation either to aspirate a lump or to explore it surgically, one should defer these until it has been studied carefully and a complete physical examination has been made. If a swelling is painless and is discovered accidentally, it very probably is neoplasm, particularly if limited to the lateral aspects of the neck. In the midline it may be either a thyroglossal cyst, dermoid cyst or an adenoma of the thyroid gland involving the pyramidal lobe or the isthmus. The presence of tenderness, pain and increase in temperature indicate an inflammatory origin. A single node or mass commonly suggests either a congenital abnormality or primary neoplasm, while a multiplicity of masses suggests lymphadenopathy.

FIG. 4.—This man developed a painless swelling on the right side of the neck in close relation to the anterior border of the sternomastoid at the level of the bifurcation of the carotid. While this case proved to be a metastatic lymph node secondary to carcinoma of the larynx, nodes in this locality commonly are observed in cases of carcinomas of the pharynx, tonsil and larynx as well as the floor of the mouth and alveolar process, also the auditory apparatus.

Early fixation, particularly if there is some tenderness, suggests an inflammatory process or tuberculosis. Primary malignant tumors or metastatic lesions usually are not fixed early; later, with the occurrence of infiltration, fixation is the rule. Changes in size too must be considered. A slow increase in size may suggest neoplasm, benign cyst or tuberculous lymphadenitis. A rapid increase in size may indicate an acute inflammatory process or hemorrhage into a pre-existing tumor.

In the case of lymphadenopathy one must consider either primary lymphoblastoma, tuberculous lymphadenitis, infectious granuloma, metastasis from malignant neoplasm or simple lymphadenitis. In diagnosis a careful systemic study, including blood studies and roentgen examination as well as a study of the upper air and food passages including the teeth and alveolar processes is necessary as it is well known that carcinoma of the nasopharynx, nasal accessory sinuses, tonsillar region, tongue and laryngopharynx may metastasize to the neck in the absence of any symptoms to direct attention to the primary tumor (fig. 4).

The size, consistency and presence of inflammatory changes are of importance diagnostically. In the young, tender nodes suggest an acute pharyngeal process. Matting together of nodes with some inflammatory change usually is evidence of a tuberculous lymphadenitis. Nontender nodes may occur in lymphosarcoma, in lymphoepithelioma or in hypertrophy of adenoid tissue and chronic tonsillitis. In the adult, the most common cause of lymphadenopathy of the neck is carcinoma of the pharynx, tonsil or larynx. It appears as a nontender movable node which later becomes multiple and fixed.

In the event that one has exhausted all of the diagnostic aids, then there is justification for excision of a node or aspiration biopsy. It is unwise to contemplate irradiation therapy until one has arrived at an etiologic diagnosis in these cases. It is equally unwise to incise a swelling without first ruling out the more common lesions.

A thyroglossal cyst or fistula is practically always in or near the midline and is attached to the hyoid bone. Diagnosis usually is not difficult. It may be confused with dermoid cyst in the midline, but this commonly is found in a lateral position. Surgical treatment is the plan of therapy in either group, care being taken in the case of thyroglossal cysts to excise a part of the body of the hyoid bone and to core out the muscles of the tongue to the foramen caecum. Simple incision of the cyst is useless. This procedure creates a draining sinus which invites infection. The use of sclerosing solutions also is to be condemned.

Branchial cleft cysts and fistula occur along the anterolateral region of the neck from the auricle to the clavicle. A majority of the fistulas have an orifice situated along the anterior border of the sternomastoid muscle in the lower third of the neck and the tract extends along the anterior border of the muscle opening into the pharynx in the region of the tonsil. Branchial cleft cysts are best treated by surgical extirpation. Careful dissection is necessary to avoid the great vessels. The fistulous tract should be dissected out, but often it is difficult to follow its course and a preliminary injection with a radiopaque substance and roentgen study is desirable. Undermining the skin and the use of multiple incisions is a helpful procedure. It usually is not necessary to open the pharynx.

Cystic hygromas of the neck present a soft doughy mass. They are most commonly observed in early childhood. Little difficulty is encountered in diagnosis, as they can be easily transilluminated and distinguished from branchial cleft cysts. Treatment often is a difficult problem. Incision with drainage is not curative and invites infection. Surgical extirpation may require extensive dissection because of infiltration about muscle sheaths and vessels. The use of sclerosing solutions may be a helpful aid to surgery. Radium and x-ray preliminary to surgical removal have been employed with some benefit.

The occurrence of adenoma of the thyroid gland may present a diagnostic problem but there should be no question concerning therapy. In the single adenoma, surgical extirpation is desirable since it is recognized that many of these become malignant.

The lymphoblastomas usually are not surgical problems although a biopsy may be necessary for diagnosis. Irradiation should be employed but the prognosis generally is not good.

Tuberculous lymphadenitis now is infrequently encountered. The nodes usually are matted together. There may be found roentgen evidence of a pulmonary lesion. Irradiation and streptomycin are of value in treatment. Surgical treatment usually is employed only if there is a draining sinus.

The treatment of carcinomatous metastasis is dependent on the site and extent of the primary lesion. As a rule irradiation is merely palliative. If secondary to carcinoma of the hypopharynx, larynx or tongue, a radical block dissection with removal of the primary lesion folowed by irradiation offers the best chance. In early nasopharyngeal carcinoma with metastasis, irradiation of the primary lesion with block dissection gives some hope.

In conclusion, any swelling of the neck should be promptly investigated and a diagnosis made. In certain of the benign lesions the time of treatment is elective, while in others it should be carried out promptly.

In the adult, the occurrence of a lump in the neck should suggest primary neoplasm of the upper air and food passages and adequate studies should be carried out promptly so that appropriate treatment may be instituted.

DISCUSSION

ROBERT E. PRIEST, M.D., Minneapolis, Minn.: About three weeks ago I received Dr. Clerf's paper and I took it home and sat down and read it carefully. I realized that it was an extremely broad presentation and that any detailed discussion of it would be impossible without being tremendously redundant. I was quite puzzled as to what I might say. I thought the matter over and decided that a discusser should either be able to clarify, which isn't necessary here, to add to, which seemed to me to be impossible, or to re-emphasize. I am going to make only two points very briefly.

One is that Dr. Clerf has emphasized that one should make a diagnosis before meddling mechanically with any kind of a swelling of the neck. One should not content himself just with physical examination, but should make use of the laboratory facilities, including blood studies, examination by an internist, roentgen ray, and whatever else may be necessary.

The second point is that all of us can remember that this paper constitutes a reservoir of tremendous importance as source for material when we have a puzzling case of a mass in the neck. I, for one, will turn to it again and again.

DR. CLERF: There is no denying what Dr. Priest has said, namely that lumps of the neck need careful investigation. That applies not only to the neck. It applies to all of our diagnostic problems, the need for careful investigation to the limit of our capabilities before we plan treatment. It is well exemplified in the neck, because the neck can be so easily examined and our errors in diagnosis may become so apparent.

PROPER USE OF ANTIMICROBIAL DRUGS IN ACUTE OTITIS MEDIA

MIRIAM H. RUTHERFORD, M.D.
OAKLAND, CALIF.

THE development and use of chemotherapeutic and antibiotic drugs during the past fifteen years has brought about new concepts in the treatment of diseases. The mortality and morbidity of diseases such as pneumonia, peritonitis, septicemia and meningitis have been greatly reduced. The results in acute otitis media have been less striking; nevertheless, too many pediatricians and general practitioners are convinced that by using these drugs alone otitis media may be cured without complications.

There is no doubt that the utilization of the sulfonamides and, more recently, of penicillin and the newer antibiotics during acute nose and throat infections has decreased the incidence of acute otitis media and its complications. Because of the apparent quick and often dramatic clinical response of many patients to antimicrobials, the opinion has become prevalent that such medical treatment alone is all that is necessary. This complete reliance upon antimicrobials has become so widespread that many physicians have discarded the time-consuming procedures of adequate history-taking and thorough examination of the ears and nose in favor of the indiscriminate administration of antibiotic drugs.

Even in large teaching centers interns and residents are not being taught the anatomy and physiology of the middle ear or a knowledge of the pathologic processes which may occur within it. The complacent acceptance of the antibiotics as a cure-all in acute otitis media has led to a state where the younger pediatricians do not know the art of cleansing the ear canal, of observing and recognizing the changes in the tympanic membrane which indicate pathologic conditions in the middle ear, or how to do a myringotomy. In fact, some of these young men and women have been taught that myringotomy is not only an unnecessary procedure but one to be avoided because it injures and scars the tympanic membrane, although spontaneous rupture of the drum under antibiotic therapy apparently is not considered harmful. They do not differentiate between suppurative and nonsuppurative acute otitis media; the treatment is always one of the antibiotic drugs without regard to the bacteriology or the pathology involved. If one drug does not bring quick results, another antibiotic is resorted to, or possibly "combined therapy," the shotgun method, is used. Such haphazard treatment could be expected to produce unsatisfactory results, and often does.

As time goes on there is growing evidence that the promiscuous use of the antibiotics and the disregard of sound surgical principles is hazardous in the treatment of otitis media. Incomplete bacteriostasis and bactericidal activity of the antibiotics results in repeated recurrences. There is ample evidence to show that the tendency to recurrences of the infection predominates in those patients who have no antibacterial antibodies and who fail to develop them

From the Department of Surgery, Division of Otorhinolaryngology, Stanford University School of Medicine, and the Department of Otorhinolaryngology, Childrens Hospital of the East Bay.

Presented at the Fifty-Seventh Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 12-17, 1952, Chicago, Ill.

during the course of the disease.[9] Natural immunity may not be acquired when the drugs are used[3] and the organisms may regain their former activity when the drug is discontinued.[7] Clinical observation certainly supports this conclusion. When acute otitis media is treated with antibiotics without myringotomy there is usually relief of symptoms but this does not indicate a cure has occurred. As Tobey[12] has pointed out, "the sulfonamides are free to act upon infection wherever they can be carried by the blood stream, therefore the most striking results occur in . . . infections such as meningitis, bacteremia and pneumonia. Wherever the blood stream is free to carry the drug, we may expect therapeutic results. The natural corollary is that wherever the blood vessels are blocked by thrombosis, as in proximity to a localized abscess, the drug is not free to act and one of the primary principles of surgery comes into play, namely, that a localized suppurative focus must be evacuated. This principle is peculiarly applicable to the ear spaces where the blood supply is meager, the vessels small and easily thrombosed and the infection enclosed in a bony cavity." When medication is discontinued too soon, infection may remain in the mucous membranes and in the exudate, consequently is able to reproduce symptoms in a short time. These recurrences are often mistaken for new infections; it is not uncommon to obtain a history of recurring earaches every few weeks or oftener over a period of months, each one being "cured" by a "shot" or two of penicillin or an ounce of terramycin.

The indiscriminate and routine use of antibiotics in acute otitis media is unwarranted and unnecessary. Such use of these powerful drugs without regard to the kind of infection present or to the condition of the drumhead and middle ear is frequently wasteful of the drugs and often obscures progression of the disease. Irreversible changes may take place while the physician is lulled into a false feeling of optimism due to the suppression of clinical symptoms by the bacteriostatic action of these drugs.

Acute otitis media is, as a rule, a self-limiting disease. Fowler[6] found that 80 per cent of unselected clinic cases got well without chemotherapy or major surgery in less than four weeks and an even higher percentage recovered if the ear canal was kept clean, the nasal mucous membranes were kept shrunk and proper care was given the eustachian tube. A still higher percentage get well if the disease is nonsuppurative. Therefore, the routine administration of antibiotic drugs to every case of otitis media is unnecessary and wasteful, since four children would be getting them needlessly to every one who may benefit. At the same time, routine administration of the drugs eliminates the classical clinical signs and symptoms which have always enabled the physician to differentiate simple, uncomplicated otitis media from those cases which would progress along a stormier course. The majority of middle ear infections which responded magically to one or two injections of penicillin or an ounce of terramycin are those which in years past responded to glycerin drops, heat and decongestants.

Nonsuppurative otitis media, observed frequently as a complication of the common cold, of viral infections of the respiratory tract, of hypertrophied adenoid tissue and of allergic rhinitis, may cause symptoms of pain, low-grade fever and hearing loss in children. Otitis media of this type is caused by obstruction of the eustachian tube and responds to decongestants which restore aeration of the middle ear and permit drainage of the secretion. Much of the evidence of the effectiveness of penicillin in the

treatment of acute otitis media is based on the false premise that otalgia means suppurative otitis media. As a matter of fact, the otalgia may be due to a reddened, retracted tympanic membrane and a vacuum in the middle ear or to an accumulation of nonsuppurative transudate or exudate. It is well established that acute nonsuppurative otitis media and most suppurative infections of the middle ear with bulging of the drum respond to myringotomy and treatment directed toward the underlying causes. If penicillin is administered at the same time, the credit for the rapid improvement is given to the drug, whereas in most cases it is the result of releasing the pressure of fluid or pus in the middle ear.

Prior to the advent of the first chemotherapeutic drugs, the complications of acute otitis media occurred fairly often in spite of myringotomy and other local and general measures. However, it has been noted by many observers that such complications occurred far more frequently in ears in which spontaneous rupture had taken place than in those in which an early myringotomy had been done. Williams,[14] in 1933, reported that the incidence of surgical mastoiditis in a large series of scarlet fever patients was 6.6 per cent where the drum membrane was incised compared to 9.3 per cent where spontaneous rupture occurred. In a later series of 427 cases of otitis media in measles, the same author[13] found that mastoidectomy was done in 4.4 per cent of cases with paracentesis, whereas 11.3 per cent of those without paracentesis required mastoidectomy. Page,[10] in a series of 836 cases of acute otitis media, reported an incidence of 5.1 per cent mastoidectomies, of which three times as many followed spontaneous rupture as followed myringotomy. Bozer[1] reported a series of 105 mastoidectomies at the Buffalo Children's Hospital during the years 1945 and 1946. Of these, 73 cases had suffered spontaneous rupture of the drum and 10 had not had *any* aural discharge up to the time of surgery. Only one case had had early myringotomy. This would seem to indicate a definite lack of understanding that the middle ear should be drained when it contains pus. Most of these cases had been treated inadequately with chemotherapeutic drugs. Richardson[11] reported an incidence of 5.3 per cent mastoidectomies out of 625 cases of otitis media treated with myringotomy over a five-year period at the Massachusetts Eye and Ear Infirmary. On the other hand, House[8] reported a series of 1,241 patients seen at the Los Angeles General Hospital during the year 1935, of which 45.9 per cent required simple mastoidectomy. In a series of 193 cases at the same hospital during the year 1943, after the therapeutic use of sulfonamides, he found an incidence of only 17.6 per cent requiring mastoidectomy. Butler,[2] in 1948, studied the results of 461 cases of purulent otitis media seen in an army clinic and treated without any chemotherapy. Out of this entire group only two patients required mastoidectomy.

It is always difficult to evaluate such statistics. The incidence of complications is usually less in private practice and in clinics than in hospitalized patients, especially those in a charity hospital who have not had the benefit of previous early care. In addition, the advent of chemotherapy has made it possible to wait longer before surgical interference is contemplated.

The fact remains that in spite of adequate care, complications sometimes did occur prior to the use of chemotherapeutic and antibiotic drugs. In pre-antibiotic times it was not an uncommon experience to have cases of acute otitis media, especially those occurring with

scarlet fever, in which the drum melted away and the patient went on to mastoiditis in spite of all efforts. In the treatment of such cases the sulfonamides and penicillin have proved to be powerful therapeutic agents. They have made it possible to avoid most of the complications of acute otitis media when used as adjuncts to local and general therapy for patients who do not respond because of the virulence of the organism, lack of immunologic response, or particular anatomic configurations. To point out that due to the indiscriminate and empirical use of antibiotics, complications are occurring now with greater frequency than they should in no way detracts from the wonderful results obtained by rational and adequate administration of antibiotics when the diagnosis of impending complications is made accurately under competent observation.

New generations of virulent organisms are arising and complications are occurring with greater frequency. Owing to antibiotic suppression, mastoiditis may develop and progress to almost complete bony breakdown without alarming symptoms. Evans[5] reported last year on 30 cases in which mastoiditis had developed in many instances without pain, fever or elevated white counts. All of these patients had been treated with insufficient doses of antibiotic drugs and none had had the benefit of myringotomy. During the same period he treated 45 cases of acute otitis media in the clinic with myringotomy and none of these developed mastoiditis. Intracranial complications are occurring in spite of antibiotics; their recognition is vastly more difficult and treatment sometimes too late because of the masking effect of the drugs. One example observed recently was a two-year-old boy who had recurrent attacks of otitis media over a period of three months. Following the fourth attack his drum healed after prolonged antibiotic therapy and remained dry. The child apparently was healthy in every respect three weeks later when he fell and struck his head. Only thirty-six hours later he was dead of a fulminant pneumococcus meningitis in spite of heroic measures to save his life. At autopsy the same organism was cultured from exudate in the mastoid of the apparently cured ear. In another case, a ten-year-old boy who was seen at home by his pediatrician had a "red ear" and some râles in the chest suggesting pneumonitis. He was given 400,000 units of procaine penicillin and the same dose was repeated the next day, following which all symptoms subsided. He was apparently well for almost three months when he appeared with a large fluctuant postauricular swelling. He had not complained of pain and had only a very low-grade fever but had noted some hearing loss which he had not mentioned. At surgery a large epidural abscess was disclosed, although the middle ear was clean since it had been sealed off at the aditus. Such cases illustrate the difficulties in the diagnosis of smoldering infections in the mastoid when suppressed by antibiotic drugs.

In the enthusiasm for the routine antibiotic treatment of otitis media to prevent mastoiditis, another complication has been lost sight of, that of chronicity. The promiscuous and inadequate use of antibiotics without myringotomy has allowed many patients to pass through the acute stage only to be left with the handicap of a chronically discharging ear with all its physical, social and economic problems. The practice, widely accepted among pediatricians and general practitioners, of allowing the ear drum to rupture when and where it will shows an appalling ignorance of the dangers of the chronically discharging ear. The site of the perforation has

a bearing upon the prognosis and, while many drums rupture where healing is likely to occur, others may perforate in Shrapnel's membrane leading to chronic suppuration in the attic with all the dangers of extension of the disease to the labyrinth, lateral sinus, facial nerve and meninges.

The incidence of hearing loss from serous effusion of the tympanum is higher than it has ever been.[4] Repeated attacks of acute secretory otitis are probably the most common cause of chronic deafness; such attacks lead to the formation of adhesions in the ossicular chain or between the tympanic membrane and the promontory, and to thickening and fibrosis of the mucous membranes. Yet in many instances these are being treated with antibiotics instead of attention being applied to the underlying cause of obstruction of the eustachian tube. Such use of the drugs shows ignorance of the pathologic processes involved although the physician may feel he is using adequate treatment throughout repeated attacks. The unwarranted supposition that penicillin cures all acute middle ear disease makes it appear unnecessary to determine by careful inspection of the tympanic membrane just what process is going on. When chemotherapy is used without myringotomy, sterile exudate may remain in the middle ear, giving rise to chronic low-grade inflammatory and adhesive changes which result in permanent hearing impairment.

In examining children referred by the schools for hearing loss it is very common to obtain a history of repeated attacks of earache but, in the words of the parents, "they all got well with penicillin and never had to be lanced." Yet most of these children have thickened, retracted tympanic membranes and a conductive hearing loss of 15 to 35 decibels. There is usually a large adenoid mass, a marked nasal allergy, chronic sinusitis or all three conditions, none of which have been corrected by the administration of penicillin.

The object of treatment in acute otitis media is not only to eradicate infection and prevent complications but to restore and preserve hearing. In the flush of excitement over the use of antibiotics to prevent mastoiditis, the function of the ear, that of hearing, has been overlooked. Most of the conductive deafness of adult life has its origin in the ear infections of childhood. Prompt diagnosis and treatment by physicians trained to observe and interpret the changes that occur in the tympanic membrane during acute infections is essential if hearing loss and other complications are to be prevented. Knowledge of the anatomy and physiology of the middle ear and eustachian tube and thorough understanding of the pathologic processes which may involve them is also essential.

In most cases of acute nonsuppurative otitis media, oral decongestants, nasal shrinkage and myringotomy, if the tympanic membrane is bulging, are indicated treatments. After the more acute symptoms have subsided, inflation of the eustachian tubes or politzerization may be necessary. If recurrent, search for the underlying cause is required.

In uncomplicated acute suppurative otitis media the pus should be drained by myringotomy, and the nose and nasopharynx cleaned and kept shrunk by oral and topical decongestants. When allergic rhinitis plays a prominent part, antihistaminics should also be used. If spontaneous rupture of the tympanic membrane has occurred and drainage is inadequate or the perforation is in Shrapnel's membrane, myringotomy should be performed in the posterior-inferior quadrant.

With good drainage, frequent cleansing of the canal and tympanic mem-

brane, and treatment of the nose and nasopharynx, recovery is rapid without antibiotics in the majority of cases of acute otitis media. In those cases in which a febrile reaction and pain persist in spite of free drainage, antibiotics should be administered. They should also be used when there is a coexistent acute follicular tonsillitis and in children who have systemic diseases such as nephritis, diabetes or rheumatic fever. When the process follows the course of normal mucous membrane suppuration, the discharge gradually changing from serosanguineous to purulent and finally to a mucoid type as the membrane becomes paler and thinner, no antibiotic drugs need to be used. At any time during the healing process antibiotic therapy may be instituted if there is recurrence of fever, pain or purulent discharge indicating unsatisfactory progress. For good results, the drug used must be effective against the organism, must be given in adequate dosage and must be continued until all infection has subsided and the ear has returned to normal. This means that not only must fever and discharge have ceased but that the tympanic membrane must be thin, pale and in normal position and that hearing must have returned to normal. If hearing impairment persists after inflation of the eustachian tubes, further investigation should be undertaken. The removal of diseased and hypertrophied adenoids, the treatment of sinus infections, irradiation of the nasopharynx in selected cases, or treatment of nasal allergy may be necessary to restore normal hearing.

In support of the foregoing concepts of treatment are results obtained in a series of 150 cases of acute otitis media in children treated in private practice during the past two years. Only those cases in which myringotomy was indicated were included; of these 36 (24 per cent) had already undergone spontaneous rupture with inadequate drainage. Six of these had ruptured while on antibiotic drugs and six others on similar treatment had bulging of the tympanic membrane. Of the entire group of 150, 108 (72 per cent) were given no antibiotic or chemotherapeutic drugs. Temperatures in this group ranged from normal to 104 degrees prior to myringotomy and were all below 101 degrees twenty-four hours later and normal on the following day. Duration of discharge was from four to fourteen days, the majority becoming dry between the sixth and ninth days. There were no recurrences and no complications.

The remaining 42 cases (28 per cent) were treated with antibiotic drugs for various indications. Eleven had persistence of fever and otalgia in spite of free drainage, five had concurrent acute follicular tonsillitis, one had had an adenoidectomy four days previously, and three had symptoms of mastoiditis. Of the latter, one was a six-year-old boy, almost comatose, with fever to 104 degrees and neck stiffness, who had been seen and had spinal puncture done in an emergency hospital earlier that day. Only after spontaneous rupture of the drum occurred and aural discharge appeared was he referred to an otologist. The remaining 22 cases (14.7 per cent) were administered antibiotic drugs later in the course of the disease because of unsatisfactory or prolonged resolution of the infectious process. The prompt response to antimicrobial drugs in these cases is very gratifying since the natural antibacterial antibodies have already reached their maximum. There were no surgical complications, no chronic otorrhea or perforations and no recurrences. Hearing returned to normal in all those patients that could be tested, although in some cases adenotonsillectomy, irradiation of the nasopharynx or

eustachian inflations were necessary in the follow-up period.

In closing I would like to quote Fowler[6] who stated that "any physician who in shot-gun fashion prescribes a drug to all cases without discrimination is reflecting ignorance, laziness or poor judgment as to the expectancies of serious trouble from otitis media." If complications and permanent hearing loss are to be prevented, it is essential that pediatricians and general practitioners, who treat most cases of acute otitis media, be made aware of the consequences of improper treatment. To accomplish this purpose the otologist must take a more active part in teaching medical students, interns and residents so that they may enter practice with a better knowledge of the anatomy and physiology of the middle ear and the pathologic processes that occur within it.

REFERENCES

1. Bozer, Herrmann E.: A study of surgical mastoiditis occurring in children at the Buffalo Children's Hospital during the years 1945 and 1946, New York State J. Med., 48:183-186 (Jan. 15) 1948.
2. Butler, Harry: Low incidence of mastoiditis in series of cases treated without sulfonamides, Ann. Otol., Rhin. & Laryng., 57:992-998 (Dec.) 1948.
3. Converse, John Marquis: Recurrence of otitic infections due to the beta-hemolytic streptococcus following inadequate sulfanilamide therapy, J.A.M.A., 113:1383-1387 (Oct. 7) 1939.
4. Eagle, Watt W.: Secretory otitis media, Ann. Otol., Rhin. & Laryng., 55:55-67 (March) 1946.
5. Evans, Maurice G.: The treatment of acute suppurative otitis media, Ann. Otol., Rhin. & Laryng., 60:638-647 (Sept.) 1951.
6. Fowler, Edmund Prince, Jr.: The bacteriology of acute otitis media and its course under new and old methods of treatment, Canad. M.A.J., 44:372-380 (April) 1941.
7. Gilmore, Goodlatte B.: Acute mastoiditis masked by treatment with sulfonamide compounds; clinicopathologic considerations, Arch. Otolaryng., 37:785-788 (June) 1943.
8. House, Howard P.: Otitis media: a comparative study of results obtained in therapy before and after the introduction of the sulfonamide compounds, Arch. Otolaryng., 43:371-378 (April) 1946.
9. Lyons, Champ, and Ganz, Robert Norton: Clinical objectives in the chemotherapy of hemolytic streptococcic infections, Arch. Otolaryng., 33:490-491 (March) 1941.
10. Page, John Randolph: Bacteriology of acute infections of the middle ear and mastoid, Arch. Otolaryng., 20:447-451 (Oct.) 1934.
11. Richardson, John R.: Observations in acute otitis media, Ann. Otol., Rhin. & Laryng., 51:804-816 (Sept.) 1942.
12. Tobey, Harold G.: The sulfonamide drugs in the treatment of acute otitis media, Ann. Otol., Rhin. & Laryng., 51: 945-954 (Dec.) 1942.
13. Williams, Horace J.: Acute suppurative otitis media in measles: a report of 427 patients, Ann. Otol., Rhin. & Laryng., 44:956-960 (Dec.) 1935.
14. ———: Otitis media in scarlet fever, Arch. Otolaryng., 17:235-242 (Feb.) 1933.

DISCUSSION

JOHN A. BIGLER, M.D., Chicago, Ill. (by invitation): A discussion of the treatment of otitis media by a pediatrician before a group of otologists can be a ticklish problem. From our viewpoint if an upper respiratory infection is properly treated, otitis media should be uncommon; mastoiditis, chronic otorrhea and deafness should be rare. This statement should hold true unless the antibiotics cannot keep pace with new types of bacterial infections or organism resistance. Our experiences at Children's Memorial Hospital are quite the opposite of those of Dr. Rutherford.

Since the general use of antimicrobials, our incidence of mastoidectomy has decreased from 114 in 1940 to 1 in 1950, and none in 1951 and so far in 1952. Our incidence of myringotomies has followed about the same pattern. Otitis media is much less frequent that formerly, I believe due to the early use of the antibiotics in children with upper respiratory infections. Most cases of otitis media and its complications that we see occur in those children that have inadequate antibiotic therapy. I mean by inadequate, no therapy or too small a dose, or antibiotics

for too short a time. It is unfair criticism to designate as failures that group of cases that have been treated for only 24 to 48 hours. That is a failure of the physician rather than that of the disease. The otitis media we are talking about is an infection like any other infection and should be treated accordingly. Who expects to treat a pneumonia or any other infection for one or two days and have a cure? Otitis media due to blockage of the eustachian tube, whether due to allergy or some other cause, is a potential bacterial otitis.

I cannot go along with the school of thought that states that the antimicrobials should be used only after conservative methods of therapy have proved inadequate. It has always seemed more logical to me to treat all cases as early as possible, with high dosage over an adequate period of time. I think failures now are due mostly to late therapy and discontinuance of therapy too soon, that is, after relief of temperature and pain.

The broad spectrum antibiotics decrease the amount of guessing we have to do as to the etiologic organism. Spontaneous rupture of the drum is now uncommon and myringotomy should be rare. With faith and confidence and the proper use of our present antimicrobials, otitis media and its sequelae should be of uncommon occurrence. This does not presuppose that resistant or new bacterial agents will not appear but our present generation of children should have the full benefit of our present knowledge.

DAVID D. DEWEESE, M.D., Portland, Ore.: I appreciate the privilege of being asked to discuss this paper.

The majority of the views expressed by Dr. Rutherford have been discussed by otolaryngologists for several years, but formal presentation of these views has long been overdue. It is, perhaps, well that they are presented first before this Academy and are thus open for general discussion. However, the message contained in the paper would be of greater value and, I do not doubt, would produce more discussion were it presented before the Academy of Pediatrics or the Academy of General Practice.

I am in strong agreement with many statements made by Dr. Rutherford. Her condemnation of the inadequacy of history taking and the lack of thorough examination of patients with otitis media is, I am convinced, entirely justified. Too often the recently trained physician will glance at an ear, then begin treatment, often without even removal of cerumen.

I most certainly agree that pediatric teaching in the last ten years has been almost unanimously "no need for myringotomy." My students tell me this. As a result, the word "myringotomy" has almost dropped out of the pediatrician's vocabulary. However, I believe there are signs of a change, even though there are still many "die-hards." It is most unfortunate that students, interns, and residents, and even more unfortunate, that parents and other lay people have been told repeatedly that myringotomy is injurious to the ear drum. I do not know of a single physician or of any scientific paper showing this to be true. As Dr. Rutherford has pointed out, there is a great deal of recorded evidence that early myringotomy (the earlier the better) not only shortens the duration of acute otitis media but is a deterrent to complications and increases the probability that hearing will be normal.

Allergic rhinitis, sinusitis, and nasopharyngeal lymphoid tissue, I agree, also, may play a part in producing either otitis media with effusion or chronicity in acute suppurative otitis media. Nevertheless, the presence or absence of these conditions does not determine whether antibiotics should or should not be used in treatment of acute otitis media. We all know that if they are neglected the course of the otitis may be prolonged and the ultimate hearing result may be poor, whether antibiotics are used or not.

I cannot agree with Dr. Rutherford that antibiotics have given less striking results in otitis media than in other diseases. In my experience the proper use of antibiotics in all cases of suppurative otitis media has totally prevented surgical complications. By this I imply that I have had none. Specifically, in twelve years of otolaryngologic practice I have never found it necessary to perform a mastoidectomy on any patient who came to me for initial treatment of the otitis. In a review of every case of acute mastoiditis requiring operation, in my private practice and in the department of otolaryngology at the University of Oregon Medical School, the significant factor making operation necessary has been either no treatment of the otitis media or undertreatment. In my opinion, "Shot gun therapy" is not nearly so much at fault as complacent undertreatment. Dr. Rutherford properly emphasizes the danger

in the complacency produced by apparent cures with "one shot treatment." We are on dangerous ground if we advocate antibiotic treatment only in 25 or 30 per cent of cases, and no antibiotic treatment in the remainder.

In our attempt to re-educate the pediatrician and the general practitioner regarding the proper treatment of acute otitis media we must emphasize adequate treatment for a long enough time to insure cure. The individual otolaryngologist is in a different position. He is specifically trained, or should be, to recognize the minor variations of history, physical signs, and other factors which make one otitis media different from another. His decision to use or withhold antibiotics often is based on this knowledge. If it is as well substantiated as Dr. Rutherford's results are, I believe he is entirely justified in his choice of treatment. The same rules, however, cannot and do not apply to the physician less specifically trained in this disease. Therefore, I should like to stress a point brought out in the paper, that is, treatment should not stop until the ear is dry, the ear drum is normal, and hearing is normal. I am firmly convinced that the mistakes of undertreatment are producing what few complications we still see. From a practical point of view, we must admit that even though four of five untreated ear drums will heal without residual, the complications that occur are serious enough to compel us to attempt to prevent all of them, even if this means treating many patients more vigorously than might be indicated on a purely statistical basis.

Meningitis and brain abscess due to pneumococcus mastoiditis and following presumed cure from antibiotics will certainly continue to be seen occasionally. This is our old friend "masked mastoiditis" which is so frequently produced by the pneumococcus type III. I recently saw a 12-year-old girl with a subperiosteal abscess, complete destruction of the inner table, and an epidural abscess lying over both the middle and posterior fossae. This was the result of a pneumococcus infection in which there had been no earache, no loss of hearing, no discharge. At the first examination the ear drum was intact, normal in color, and without evidence of disease. The pathologic findings, described above, were present at operation the following morning. Certainly, inadequate treatment cannot be blamed for some victims of this notorious killer.

Finally, I agree with Dr. Rutherford that we should continue to make a stronger plea for myringotomy. A survey of 268 consecutive, unselected cases of acute otitis media (studied for the purpose of this discussion) indicates that of 26 patients who showed some loss of hearing following resolution of the infection, 22 had had what appeared to be adequate spontaneous rupture and only 4 had had myringotomy. The ratio of apparent adequate rupture to myringotomy was approximately equal. Although this is a small series, it strongly suggests that our best hearing results will be obtained if we continue to advocate myringotomy in addition to adequate chemotherapy.

Again, I wish to thank the secretary of the Academy for the privilege of discussing this paper and to compliment Dr. Rutherford on a fine presentation.

MIRIAM H. RUTHERFORD, M.D., Oakland, Calif.: First of all, I should like to thank Dr. Bigler and Dr. DeWeese for discussing this paper.

I agree with Dr. Bigler that the treatment of acute respiratory infections with antibiotics has reduced the incidence of complications, including acute otitis media. I do believe that otitis media is less common now than it was fifteen years ago. The fact remains, however, that even with the use of antibiotics acute otitis media does occur and that in any event not every child with an acute respiratory infection gets antibiotics in the early stages.

The use of antibiotics in insufficient amounts is not a geographic idiosyncrasy but is common everywhere. Nor is such use related to climate. Such practices, as noted in my paper, also occur in Boston which is quite distant from Oakland and has a distinctly different climate. The insufficiency of antibiotic treatment and the dependency upon it to the exclusion of local treatment and drainage is common among general practitioners and pediatricians throughout the country. These physicians tend to regard elimination of pain and fever as having effected cures. These are the practices that are objectionable because they lead to complications of mastoiditis, brain abscess, and so on. Furthermore, it leads to a much more common but less dramatic complication, that of hearing loss. This complication comes to the attention of the otologist rather than the pediatrician. An alarming number of such cases are being revealed by hearing surveys in schools.

Dr. DeWeese feels that every child with otitis media should be treated with penicillin or one of the antibiotic drugs. There is no objection to this if, at the same time, the physician examines the ear, keeps it under observation and drains exudate as may be indicated. My point was that antibiotics need not be used in every case. Another important point is the lack of knowledge of how to look at ears and ear drums and how to evaluate what is going on within the middle ear. This deficiency and blind faith in antibiotics I believe is responsible for so many instances of impaired hearing in children now.

I do not agree with Dr. DeWeese that complications may always be prevented by early use of antibiotics because I have seen too many children on adequate doses of antibiotic drugs develop bulging ear drums followed by spontaneous rupture. I believe it is essential that myringotomy be taught to those who are going to treat acute otitis media.

A STRONGER MAGNIFIER FOR ENDOSCOPES

WILLIAM T. K. BRYAN, M.D.
ST. LOUIS, MO.

THE VIEW one obtains with an endoscope is generally inferior to that seen in good photographs and movies. The difference lies largely in the magnification, and although some improvement is obtained with a 2× magnifier such as Broyles', I felt higher magnification, such as 7× or more, would be better. A second desideratum was the possibility to use the usual instruments, as suction tips and forceps, with the magnifier. Thirdly, the magnifier should be easily brought in and out of the line of vision. A magnifier incorporating these features is shown in the illustrations (figs. 1, 2 and 3).

For magnification greater than 2 to 4× it is customary to use a compound system which includes a compound ocular, an erecting system which usually consists of two right-angle prisms, and an acromatic lens for the objective. Because of its availability and adaptability we made use of one side or half of a 7 by 18 mm. binocular. The necessary short focus was obtained by introducing an extension to the ocular sleeve. The optical system was then attached to the handle of a laryngoscope by means of a clamp, rod, and hinge.

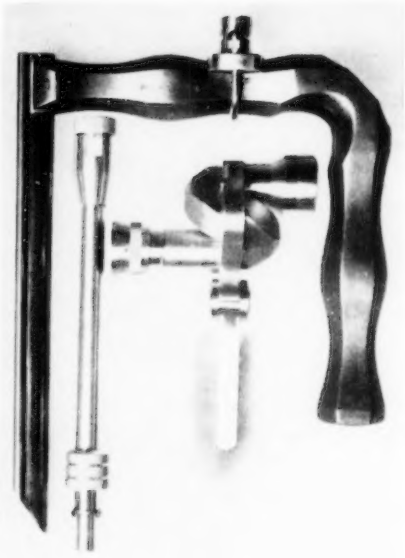


FIGURE 1.

In order to use the magnifier with a bronchoscope or esophagoscope, it was necessary to modify the clamp and to extend the focal distance by use of a supplementary lens. A cap was then made to fit over the objective to carry both the supplementary lens and a piece of plane glass to protect the lens.

Presented as a *New Instrument* at the Fifty-Seventh Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 12-17, 1952, Chicago, Ill.

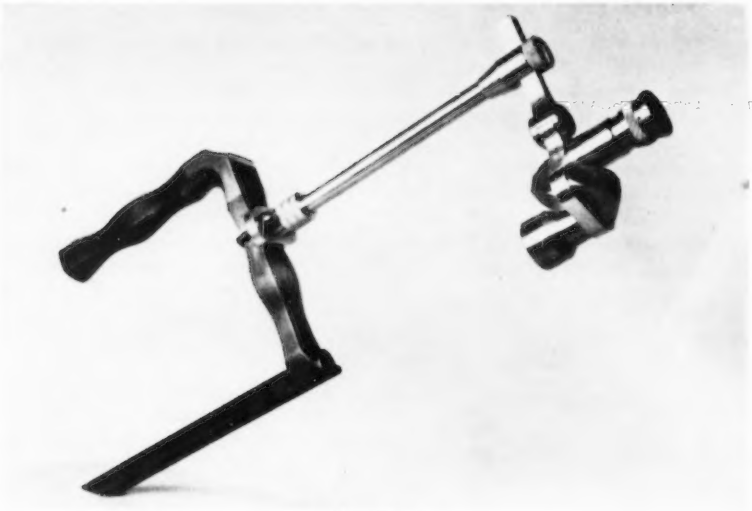


FIGURE 2.

In using higher magnification, more light was necessary, so a second light carrier was built into the laryngoscope. Fogging of the lens is avoided by warming the instrument before use.

After using the magnifier, I can report that it is not seriously in the way and that it has helped in the accurate removal of benign vocal cord tumors. Patients' movements, of course, are likewise magnified, and are just that much more disturbing. The same is true of bleeding. Nevertheless, when these conditions are under control, the view is excellent and instrumentation is refined.

FIGURE 3.

CORTISONE AND HYDROCORTISONE IN OCULAR INFECTIONS

PHILLIPS THYGESON, M.D.
SAN JOSE, CALIF.
MICHAEL J. HOGAN, M.D.
and
SAMUEL J. KIMURA, M.D.
SAN FRANCISCO, CALIF.

CORTISONE, and more recently hydrocortisone, have come into wide use in ocular inflammations due to a variety of causes. Their therapeutic effects have been most striking and useful in inflammations of a strictly allergic nature, such as hay-fever conjunctivitis and vernal catarrh; in the ocular manifestations of the so-called collagen diseases, such as lupus erythematosus; and in bacterial diseases, such as phlyctenular keratitis and nongranulomatous uveitis, in which the allergy of infection is believed to be the main cause of the ocular disease. On the other hand, such bacterial infections as pneumococcic or Koch-Weeks conjunctivitis, in which the hypersensitivity factor is minimal, have with few exceptions been unresponsive, and it is now known that a number of infectious agents can be made more destructive by the systemic use of cortisone. This has been most apparent in viral diseases, such as herpes simplex, poliomyelitis and trachoma, but has also been observed experimentally in tuberculosis, syphilis and streptococcic infections. Nevertheless, the hormone has been used with apparent success in conjunction with specific antibacterial therapy in certain systemic infections, such as typhoid fever,[1,2] peritonitis[3,4] and pelvic cellulitis,[5-7] in which a toxic factor was particularly prominent.

The confusion surrounding the use of cortisone in infections indicates a need for further clinical and experimental investigation into the conditions under which the nonspecific, anti-inflammatory effect of the hormone can be applied favorably to ocular infections, either with or without specific anti-infectious therapy. The cornea seems to be an ideal tissue on which to study this effect because of the possibility of producing standard corneal ulcers with a variety of bacteria, and because of the ease with which infiltrative and destructive corneal changes can be observed. For these reasons the cornea was chosen in preference to other tissues for this clinical and experimental survey.

The study is divided into the following three parts: I. A review of the literature on cortisone in infections in general and ocular infections in particular. II. A clinical study of the effect of cortisone and hydrocortisone on corneal ulcers of known and unknown origin. III. An experimental study of cortisone and hydrocortisone in bacterial corneal infections.

I

THE EFFECT OF CORTISONE ON INFECTIONS

The manufacturers of ACTH and cortisone have made no claims that

From the Department of Ophthalmology and the Francis I. Proctor Foundation for Research in Ophthalmology, University of California School of Medicine, San Francisco.

Presented at the Fifty-Seventh Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 12-17, 1952, Chicago, Ill.

their products act beneficially on any specific bacterial or viral infection; in arthritis, moreover, in which results have in general been most spectacular, the hormones are considered to be contraindicated in the infectious types due to tuberculosis, gonorrhea, etc. Nevertheless there has accumulated a considerable literature concerned with the favorable and unfavorable effects of both preparations on experimental and clinical infections of different types, and with the mechanisms of these effects.

The Effect of Cortisone on Tissue Resistance and the Mechanism Of Immunity

Effect on bacterial allergy. Cortisone administered systemically is known to inhibit the tuberculin reaction[51] and to diminish or abolish signs of bacterial allergy in certain other diseases. Perhaps the most striking example of this inhibition occurs in phlyctenular keratoconjunctivitis[54] in which the allergic ocular manifestations can be abolished rapidly by topical cortisone administration.

Effect on antibody production. In spite of initial reports of a drop in antibody titer after systemic administration of cortisone in rabbits immunized with crystalline egg albumin, numerous observers have since found the antibody titer to be insignificantly affected in comparable experiments.[4]

Suppression of the Shwartzman phenomenon. The suppression of this phenomenon has been noted by several observers, among them Marcus and Donaldson.[30]

Effect on lymphatic tissue. Cortisone is known to have caused involution of the thymus, spleen, and lymph nodes in the experimental animal.[52]

Inhibition of reactivity of connective tissue. In the normal rabbit cortisone in high dosage delays the development of granulation tissue in wounds and of new bone formation after fractures.[39] This effect has not been conspicuous clinically in man, and many examples of good wound healing after surgery in patients treated with cortisone are on record. There is general agreement, however, that corneal vascularization can be inhibited by topical application of cortisone.[20] The effect of this inhibition on acute corneal infections is minimal but in sluggish infections it may be important.

Effect on circulating eosinophils. Cortisone systemically given causes a temporary drop in circulating eosinophils, and a reduction in local cosinophilia when used topically in allergic conjunctivitis or rhinitis. No significance has been attached to this finding insofar as infection is concerned.

Effect on neutrophils. Local infiltration with polymorphonuclear leukocytes is reduced by cortisone.

Effect on sedimentation rate. The sedimentation rate is generally lower after the administration of cortisone.

Inhibition of hyaluronidase production. The inhibition of hyaluronidase production by certain bacteria has been described as a cortisone effect. Since this enzyme is concerned in the invasiveness of these bacteria, its inhibition should have an important protective effect. Experimental evidence on this point is not yet available.

Effect on nonspecific inflammation. Cortisone has been widely advocated for use in "inflammatory eye disease," no doubt by reason of its nonspecific anti-inflammatory action. It is known to block the inflammation produced by such agents as horse serum and killed streptococci in sensitized animals, and by jequirity infusion and trauma.[60,61] This remarkable ability of cortisone to modify tissue response to various agents, including infectious agents, has opened the door to its wide use as an adjunct to specific anti-infectious therapy, and fa-

TABLE I

THE INFLUENCE OF CORTISONE ON EXPERIMENTAL GENERAL INFECTIONS

OBSERVER	INFECTION	EXPERIMENTAL ANIMAL	RESULT
	Bacterial infections:		
Michael et al.[32]	Tuberculosis	Rat	Infection aggravated
Turner and Hollander[57]	Syphilis	Rabbit	Infection aggravated; rebound phenomenon
Kligman et al.[24]	Staph. aureus	Guinea pig	Infection aggravated
Mogabgab et al.[33]	Group A hemolytic Strep.	Rabbit	Bacteremia and death from intracutaneous infections normally localized
Robinson[44]	Pneumococcus	Rabbit	Infection aggravated
Glaser et al.[15]	Pneumococcus	Mouse	Survival time reduced in cervical adenitis and pneumonia
Abernathy[1]	Brucella	Mouse guinea pig, rabbit	Infection aggravated
	Fungus infections:		
Kligman et al.[24]	Trichophyton mentagrophytes	Guinea pig	Infection aggravated
Redaelli et al.[42]	Coccidioides immitis	Rat	Infection aggravated; large, necrotic, exudative foci
	Viral infections:		
Shwartzman and Fisher[48]	Poliomyelitis	Hamster	Infection aggravated
Shwartzman[47]	Poliomyelitis	Mouse	Infection aggravated; cells outside central nervous system became susceptible
Kilbourne and Horsfall[22]	Coxsackie virus	Mouse	Adult mice, ordinarily insusceptible, became susceptible
Ormsby et al.[37]	Herpes simplex	Rabbit	Infection aggravated
Kligman et al.[24]	Vaccinia	Guinea pig	Infection aggravated
Kimura et al.[23]	Vaccinia	Rabbit	Infection aggravated

vorable results have been reported clinically. To be decided, however, with respect to infection in general and to specific disease in particular, is whether inflammation is protective or harmful, and whether or not the apparently beneficial effect of cortisone is in fact a masking phenomenon only.

Inhibition of fever and toxemia. Many observers have noted the suppression of the toxic and febrile signs of disease in both experimental and clinical infections, even when the causal bacteria were undergoing rapid multiplication and dissemination. This effect was well shown by Germuth and co-workers[14] in

cortisone-treated rabbits in which the febrile response to pneumococci and heat-killed suspensions of typhoid bacilli was inhibited. Treated rabbits were furthermore protected against the acute lethal effect of a strain of Staphylococcus aureus.

Effect of Cortisone in Systemic Infections

Unfavorable effects have in general accompanied the systemic use of cortisone in infectious disease, not only in bacterial disease but in viral and mycotic disease as well. It has been shown repeatedly, for example, that the course of experimental tuberculosis[5,32] is aggravated by the administration of cortisone. Other experimental infections that have been worsened by cortisone therapy are listed in table I. It is interesting that the toxic effects of infections may be abolished at the very time the bacteria concerned are multiplying and disseminating. Thus, in the experimental beta hemolytic streptococcic infection of the cortisone-treated rabbit, reported by Mogabgab et al., the animals remained in good condition, in spite of bacteremia, until shortly before death. With respect to experimental viral infection, Shwartzman and Fisher found that cortisone aggravated significantly the infection of hamsters with two strains of poliomyelitis virus; in parallel experiments the infection was unaffected by ACTH. Shwartzman was able to produce a violent and uniformly fatal poliomyelitis infection by the intraperitoneal inoculation of this animal when it had been treated with cortisone while untreated or ACTH-treated animals were totally resistant to inoculation by this route. The virus apparently was able to multiply in tissues outside the central nervous system under the influence of cortisone.

This enhancement by cortisone of susceptibility to infection appears to be more dramatic with viruses than with other microbic agents. The enhancing effect can be explained in bacterial infection as a result of interference with the early inflammatory reaction but this phenomenon is less important in viral disease. No explanation has as yet been offered for the cell modification which occurs under the influence of cortisone and transforms cells that are normally resistant to certain viruses into fully susceptible cells.

Effect of Cortisone in Ocular Infections

Experimental infections. In comparison with the relatively extensive literature dealing with the cortisone treatment of clinical ocular infections, the literature on cortisone-treated experimental ocular infections is still scant. Table II outlines the essential findings to date. It will be noted that the effects vary from the slightly favorable effect observed by Leopold and his colleagues in experimental corneal ulcers due to Aerobacter aerogenes, Proteus vulgaris, Escherichia coli, Diplococcus pneumoniae, type 1, and Staphylococcus aureus, to the markedly unfavorable effect noted by Woods and Wood in experimental ocular tuberculosis. In experimental viral infections of the eye the results have been uniformly unfavorable.

Clinical infections. In clinical infections cortisone has been used most extensively, both systemically and topically, in various types of uveitis. Nongranulomatous iridocyclitis, which is generally believed to represent in most instances an allergic response to a focal infection remote from the eye, has been most uniformly responsive, probably as a result of the blocking of the allergic inflammation rather than of any effect on the causal focus itself. The response of phlyctenular keratitis is analogous.

TABLE II

THE INFLUENCE OF CORTISONE ON EXPERIMENTAL OCULAR INFECTIONS

YEAR	OBSERVER	INFECTION	EXPERIMENTAL ANIMAL	RESULT
1951	Leopold et al.[26,40]	Corneal ulcers produced by A. aerogenes, E. coli, and P. vulgaris	Rabbit	Drop-treated eyes similar to control eyes; eyes treated subconjunctivally less severely affected
1951	Leopold et al.[26,40]	Herpes simplex virus keratitis	Rabbit	Treated eyes involve more severely than controls
1951	Hallett et al.[16]	Herpes simplex virus keratitis	Rabbit	Keratitis aggravated by subconjunctival cortisone
1951	Thygeson et al.[55]	Herpes simplex virus keratitis	Rabbit	Keratitis aggravated by topical and systemic cortisone; results more severe after systemic treatment
1952	Woods and Wood[60,61]	Tuberculous uveitis	Rabbit	Course altered unfavorably by topical and parenteral cortisone and hydrocortisone
1952	Lepri[28]	Pneumococcal keratitis	Rabbit	Duration and sequelae unaffected by cortisone alone; simultaneous cortisone and antibiotic shortened course and attenuated inflammation
1952	Ormsby et al.[37]	Herpes simplex virus keratitis	Rabbit	Course unaffected by topical cortisone; systemic cortisone prolonged acute phase
1952	Kimura et al.[23]	Vaccinia virus keratitis	Rabbit	Topical cortisone ineffective; keratitis aggravated by systemic cortisone

On the other hand, in granulomatous uveitis due to syphilis, tuberculosis, and other diseases in which the organisms are believed to be present in the eye, the effect of cortisone therapy has varied greatly and systemic therapy is believed by many observers to be contraindicated. In the granulomatous uveitis of still unknown etiology, sympathetic ophthalmia, the response to cortisone therapy has generally been good. This is of special interest in view of the hypersensitivity to uveal pigment which often occurs in this disease.

In interstitial keratitis due to congenital syphilis, reports of cortisone therapy have been generally enthusiastic, but the treated disease, unlike the untreated disease, tends to recur.

With regard to corneal ulcers, the preliminary report to the Medical Research Council of Great Britain by the Panel on the Ophthalmological Applications of Cortisone and ACTH (Duke-Elder et al.[10]) states that "in general the response of infective keratitis, ulcers, hypopyon ulcers, corneal abscesses, and similar conditions, is good. Only one case (out of 14) failed to show any response and in three the result was equivocal." Unfortunately the etiology of these cases is not stated. These observers reported poor results in 2 cases of marginal corneal ulcers but excellent results in 2 cases of phlyctenular keratitis and in 12 cases of rosacea keratitis. As will be seen in table III, these results disagree with most of the clinical re-

TABLE III

HYPOPYON ULCERS TREATED TOPICALLY WITH CORTISONE

YEAR	OBSERVER	NUMBER OF CASES	RESULT
1950	Desvignes et al.[9]	3	Favorable
1950	Arruga[2]	1	Unfavorable
1951	Duke-Elder et al.[10]	14	Healing in 10; equivocal results in 3; failure in 1
1951	Leopold et al.[26,40]	3	All improved
1951	McLean et al.[31]	1	No improvement
1951	Raiford[41]	3	Rapid healing
1951	Thorpe[53]	6	4 improved by ACTH; 2 unimproved by topical cortisone
1952	Woods[59]	1	Condition aggravated
1952	Ormsby et al.[38]	4	Cortisone with antibiotic helpful in controlling iritis and discomfort

TABLE IV

PHLYCTENULAR KERATITIS TREATED TOPICALLY WITH CORTISONE AND HYDROCORTISONE

YEAR	OBSERVER	NUMBER OF CASES	RESULT
1951	Steffensen et al.[50]	2	Relief of pain in 2 days; complete clearing in 17 and 21 days
1952	Steffensen et al.[49]	7	Good response (hydrocortisone)
1951	Barrios and Barriere[3]		Favorable
1951	Thygeson and Fritz[54]	26	Rapid healing in 25
1951	Arruga[2]		Favorable
1951	Scheie et al.[46]	3	Remissions in 2; improvement in 1
1951	Fitzgerald et al.[12]	4	Complete recovery
1951	Leopold et al.[26,40]	1	No improvement
1951	Duke-Elder et al.[10]	2	Rapid and dramatic healing
1951	McLean et al.[31]	1	Good response
1951	Ikui[18]	2	Excellent response
1951	Von Sallmann[58]	9	Rapid healing
1951	Franceschetti et al.[13]	1	Good response
1952	Ormsby et al.[38]	1	Complete remission
1952	Thorpe[53]	2	Improvement in 1; no improvement in 1
1952	Thygeson et al.[56]	11	Uniformly favorable results
1952	Fine and Goodwin[11]	1	Healing

TABLE V

MARGINAL ULCERS TREATED TOPICALLY WITH CORTISONE

YEAR	OBSERVER	NUMBER OF CASES	RESULT
1951	Steffensen et al.[50]	1	Healing in 2 days
1952	Steffensen et al.[49]	2	Good response (hydrocortisone)
1951	Arruga[2]		Favorable
1951	Mosher[35]	1	Good response
1951	Brückner[7]		Favorable
1951	Scheie et al.[46]	6	Remission in 5
1951	Fitzgerald et al.[12]	1	Prompt recovery; recurrence
1951	Leopold et al.[26,40]	3	All improved
1952	Ormsby et al.[38]	8	All improved; relapse in 5
1952	Franceschetti et al.[13]	2	Good response
1952	Thorpe[53]	8	Good response in 6; no response in 2
1952	Fine and Goodwin[11]	5	Effective in all

ports to date on hypopyon and marginal ulcers but are in agreement with other reports on phlyctenular keratitis and rosacea keratitis.

Cortisone has indeed been extraordinarily successful in the control of phlyctenular keratitis. Acute uncomplicated attacks can be aborted within 48 hours. In cases secondarily affected by Staphylococcus aureus the cortisone has had little effect on the bacterial infection which has required adjunctive antibiotic therapy. Table IV outlines the findings reported to date.

Except for the Duke-Elder report, there has been quite general agreement (Table V) that marginal corneal ulcers are responsive to hormonal therapy. This type of ulcer is generally considered to be toxic or allergic and its lesions to be bacteria-free.

In dendritic ulcers the literature (table VI) is confused as to the effect of hormonal therapy. Favorable results have been claimed by a number of observers but the experimental studies of Hallett et al., Ormsby et al., and Thygeson et al. leave little doubt that the cortisone effect is essentially a masking one and not actually beneficial. As will be reported later, this masking effect can lead to unusual complications, including severe uveitis and corneal perforation.

The literature (table VII) contains no instance of therapeutic success with cortisone in Mooren's ulcer.

With respect to rosacea keratitis there has been quite general agreement (table VIII) as to the favorable effect of the hormone.

There seems to be no doubt from the literature that cortisone has a blocking effect on the inflammatory reaction of most corneal ulcers, but Woods[59] warns that a masking effect is to be feared. He cites a case of corneal abscess which on admission to the hospital showed conjunctival inflammation and chemosis and beginning corneal vascularization. The patient was placed on hormonal therapy immediately. Partial clearing of the inflammatory reaction and of the chemosis

TABLE VI

DENDRITIC ULCERS TREATED TOPICALLY WITH CORTISONE

YEAR	OBSERVER	NUMBER OF CASES	RESULT
1950	Olson et al.[36]	1	Good response
1951	Steffensen et al.[50]	2	Subjective improvement only; iodine cauterization required
1951	Lerner[29]	1	Improvement
1951	McLean et al.[31]	1	Good response
1951	Leopold et al.[26,40]	4	Improvement in 3; no improvement in 1
1951	Raiford[41]	2	Healing in 4 days
1952	Ormsby et al.[38]	20	All failures
1952	Thorpe[53]	2	Aggravation in 1; no response in 1
1952	Fine and Goodwin[11]	1	Doubtful effect
1952	James et al.[21]	4	Subjective improvement only; all required iodinization
1952	Moser[34]	2	No improvement; hypopyon keratitis occurred as complication in one case

TABLE VII

MOOREN'S ULCER TREATED TOPICALLY WITH CORTISONE

YEAR	OBSERVER	NUMBER OF CASES	RESULT
1951	Lavery et al.[25]	1	No response
1951	Duke-Elder et al.[10]	3	Temporary regression of vascularization in 2; symptomatic relief only

TABLE VIII

ROSACEA KERATITIS TREATED TOPICALLY WITH CORTISONE

YEAR	OBSERVER	NUMBER OF CASES	RESULT
1950	Lepri[27]		
1951	McLean et al.[31]	1	Good response
1951	Steffensen et al.[50]	1	Symptom-free after 3 days
1951	Olson et al.[36]	1	Complete remission with mild recurrence; recurrence controlled by additional cortisone
1951	Fitzgerald et al.[12]	4	Spectacular recovery
1951	Rouher[45]	1	Healing in 4 days
1951	Franceschetti et al.[13]	1	Good response
1952	James et al.[21]	2	Excellent results

TABLE IX

CORNEAL ULCERS TREATED TOPICALLY WITH CORTISONE
(THYGESON AND HOGAN)

TYPE OF ULCER	NUMBER OF CASES	RESULT
Hypopyon ulcers	3	Unimproved
Marginal ulcers	23	Rapid healing
Mooren's ulcer	2	Temporary improvement
Phlyctenular keratitis ulcer	11	Dramatic healing
Dendritic ulcers	13	Unaffected or aggravated; some subjective improvement
Marginal ulcers in rosacea	2	Dramatic healing
Rosacea keratitis	1	Rapid healing
Marginal ulcer in vernal catarrh	1	Dramatic healing

ensued but the abscess itself progressed. When the cause of the infection was determined and specific therapy instituted, regression and healing took place. Woods stressed the fact that the inflammatory, neovascularizing, and fibroblastic processes are reparative and should not be interfered with unless other adequate specific therapy is available.

II

A CLINICAL STUDY OF THE EFFECT OF CORTISONE ON CORNEAL ULCERS

In this clinical study only topical cortisone and hydrocortisone have been employed. The cortisone has been used in both the 0.5 per cent and the 2.5 per cent concentrations and the hydrocortisone, with a few exceptions, in the 0.5 per cent concentration only. They have been used only exceptionally in ointment form and then only for night application. The ophthalmic suspension was administered from four to eight times daily but usually six times. The results in the treatment of 56 cases of corneal ulceration are summarized in table IX.

Hypopyon ulcer. In all three of the cases of hypopyon ulcer, all of which were due to pneumococci, the disease progressed during the first 36 hours of cortisone therapy. At this time antibiotic therapy with terramycin was begun and was successful in arresting the disease in each case. The topical use of cortisone 2.5 per cent seemed to have little or no effect on the inflammatory phase of the disease, which was severe. In future cases it is planned to employ the antibiotic and cortisone simultaneously. This decision is based on experience with experimental pneumococcic ulcers, to be described later, in which cortisone did not interfere with antibiotic activity.

Marginal ulcer. In contrast to the lack of success with hypopyon ulceration, the success with marginal ulceration was striking, the ulcers healing rapidly within 24 to 48 hours. In the light of previous experience with this type of ulcer there seemed also to be a definite reduction in the amount of scarring. The cortisone effect appeared to be as good in the cases secondary to staphylococcic blepharoconjunctivitis as in the few occurring without gross conjunctival or lid margin disease.

A single case of marginal ulcer, which was first thought to be catarrhal in type

but later identified as herpetic, became worse under hydrocortisone therapy.

A single case of recurrent severe ring ulcer, in which bacteriologic findings were negative, responded rapidly to hydrocortisone used in ointment form. A second recurrence some months later responded in the same way.

An unusual case of bilateral marginal ulceration, resembling the Mooren type by virtue of its sluggish course and the excavation of limbal tissue, showed no response whatsoever to topical cortisone therapy. Cultures and scrapings were negative but healing occurred after topical antibiotic therapy.

Mooren's ulcer. Two atypical cases of Mooren's ulcer were seen in consultation. In the first, cortisone therapy resulted in a diminution of the inflammatory signs but the disease progressed at the usual rate. In the second case the ulcer appeared cleaner on cortisone therapy but the lesion progressed as usual.

Phlyctenular keratitis. In the 11 cases of phlyctenular keratitis the cortisone effect was dramatic. In the last 3 cases hydrocortisone was used. It seemed to be at least as effective as cortisone. In each of the 11 cases the inflammatory signs subsided within 24 to 48 hours and open ulcers epithelialized with surprising rapidity. Unlike the findings in the larger, previously reported series (Thygeson and Fritz), there was not a single refractory case.

Dendritic ulcer. Eleven cases of dendritic ulcer were treated topically with cortisone, and 2 were treated with hydrocortisone. In no case did the use of these hormones result in healing, and in 2 cases treated at onset with cortisone the disease became unusually severe, progressing to severe disciform keratitis and accompanied by a destructive iridocyclitis and, in one case, by secondary glaucoma. Both of these cases were more severe than any seen before by either of the writers, and their severity is interpreted as a cortisone effect. A definite masking of symptoms was noted, most patients stating that they felt better on cortisone even though the keratitis was objectively unimproved.

In the late, trophic stages of the disease, on the other hand, the hormones appeared in 8 cases to have a definitely beneficial effect, both subjectively and objectively. This was especially marked in old, vascularized corneas with persistent epithelial edema and a tendency to epithelial breakdowns without dendritic figures.

Rosacea with keratitis. Three cases of rosacea with keratitis were treated with cortisone. In 2 the keratitis was in the form of marginal ulceration identical in appearance with the type so commonly seen in staphylococcic blepharoconjunctivitis. In our opinion this is not a rosacea keratitis but simply a catarrhal ulcer resulting from the superimposed staphylococcic infection so common in rosacea. The third case had a vascularizing keratitis involving the lower third of the cornea. All 3 cases responded readily to cortisone therapy and at the time of writing have not recurred.

Vernal catarrh with marginal ulcer. A single case of marginal ulcer associated with vernal catarrh showed a dramatic response to hydrocortisone. The ulcer was accompanied by the typical epithelial keratitis of vernal catarrh; the keratitis disappeared on hydrocortisone therapy also. The presence of a secondary staphylococcic infection suggested the possibility that this ulcer was of bacterial origin, comparable to the ordinary catarrhal ulcer, rather than a true vernal ulcer.

III

EXPERIMENTAL STUDIES

The lack of suitable clinical material with which to assay the effect of corti-

sone on hypopyon ulcers made experimental studies mandatory. Intracorneal injection into the guinea pig of measured amounts of a bacterial suspension was found to yield suitable corneal infections sufficiently uniform to permit comparative studies to be made. The studies were designed for the purpose of determining (1) whether systemic or topical cortisone therapy promoted or retarded the course of the experimental infection, and (2) whether such therapy interfered with or promoted antibiotic action.

Experiment 1. *The Effect of Cortisone On Ulcers Produced Experimentally With Staphylococcus aureus*

Materials and methods. A twenty-four-hour culture on blood agar of a strain of Staphylococcus aureus, coagulase and mannitol positive, isolated from a case of severe blepharoconjunctivitis, was used to prepare a suspension in normal saline standardized to contain 300,000 organisms per milliliter. Both eyes of 16 guinea pigs were inoculated intracorneally with 0.1 ml. of the suspension. They were then divided into two groups, A and B, of 8 animals each.

Treatment. Group A. The 8 animals in this group received one drop of cortone suspension (5 mg. per cubic centimeter) in the right eye hourly during the working day (8 a.m. to 5 p.m.). Simultaneously the left eyes were given one drop of the cortone suspending agent. At 4:30 p.m. on the first day of the experiment the right eyes were given 0.05 ml. of the cortone suspension subconjunctivally and the left eyes were given the same dosage of the suspending agent.

Group B. The 8 animals in this group received one drop of cortone suspension and one drop of terramycin ophthalmic solution hourly in the right eye. Simultaneously the left eye was given the terramycin solution only. At 4:30 p.m. the right eyes were given 0.05 ml. of cortone subconjunctivally and at the same time each pig was given 25 mg. terramycin orally. No further cortone was given but the terramycin was repeated daily, in both eyes and orally.

Results. First postinoculation day. All eyes of the Group A animals developed abscesses which varied slightly in degree of severity. The eyes of the animals of Group B did not develop abscesses but the corneas showed nebulous opacification varying from slight to moderate degree. All the blood agar cultures from the animals of Group A showed moderate (50 colonies per plate) to heavy (300 colonies per plate) growth of Staphylococcus aureus. Cultures from the eyes of the animals of Group B ranged from no growth to heavy growth on one plate.

Second postinoculation day. The abscesses of the Group A animals were all perceptibly worse. The corneal haze of the eyes of the Group B animals had diminished. Cultures from the eyes of the Group A animals ranged from 200 colonies per plate to confluent growth.

Third postinoculation day. All the corneas of the Group A animals were necrotic. The corneas of five of the Group B animals were entirely clear, two animals had nebulous infiltrates but in the right corneas only, and one still had them in both corneas. Cultures from the eyes of the Group A animals ranged from moderate growth to overgrowth; cultures from the Group B eyes ranged from no growth to four colonies.

Fourth postinoculation day. Group A animals all showed corneal necrosis varying slightly in intensity. Group B animals showed uniformly clear corneas. Cultures from Group A eyes showed heavy growth to overgrowth. Cultures from Group B animals showed no growth in six cases to 16 colonies in one case.

Summary, Experiment 1. Neither cortisone nor the suspending vehicle ap-

peared to have any protective or deleterious effect on the course of the otherwise untreated corneal infections of Group A which went on to loss of the eyes in all animals. On the other hand, cortisone did not appear to aid or damage the protective effect of terramycin in the Group B series.

Experiment 2. The Effect of Cortisone On Ulcers Produced Experimentally With Pseudomonas aeruginosa

Materials and methods. A twenty-two-hour culture on blood agar of a strain of Pseudomonas aeruginosa isolated from a severe corneal ulcer, containing approximately 100 million organisms, was used to prepare a suspension in normal saline standardized to contain 25,000 organisms per milliliter. Both eyes of 16 guinea pigs were inoculated intracorneally with 0.05 ml. of the suspension. They were then divided into two groups, A and B, of eight animals each.

Treatment. Group A. The right eyes of the eight animals of this group received one drop of cortone suspension (5 mg. per cubic centimeter) hourly during the working day (8 a.m. to 5 p.m.). Simultaneously the left eyes received one drop of the cortone suspending agent. At 4:30 on the first day of the experiment the right eyes were given 0.05 ml. subconjunctivally of cortone suspension, and the left eyes were given the same amount of the suspending agent.

Group B. The right eyes of the eight guinea pigs of this group received one drop of cortone suspension and one drop of polymyxin B hourly on the first day. At the same time the left eyes received only the polymyxin B. At 4:30 p.m. the right eyes were given 0.05 ml. of cortone subconjunctivally and at the same time each pig was given 50 mg. polymyxin B by mouth. No further cortone was given but the polymyxin B was continued daily, in both eyes and orally.

Results. First postinoculation day. All eyes of Groups A and B showed reactions ranging from gray, patchy infiltrates to definite abscesses, and all cultures from both groups showed moderately heavy to very heavy growth on blood agar plates.

Fourth postinoculation day. All eyes of both groups grew steadily worse until the fourth day when they had all become completely necrotic. At this time all cultures from both groups were overgrown with Pseudomonas aeruginosa.

Summary, Experiment 2. Neither cortisone nor polymyxin B had the slightest effect on the course of the disease produced by intracorneal injection of Pseudomonas aeruginosa.

Experiment 3. The Effect of Cortisone on Ulcers Produced Experimentally with Beta Hemolytic Streptococci

Materials and methods. A nineteen-hour culture on blood agar of a strain of beta hemolytic streptococcus isolated from a case of conjunctivitis was used to prepare a suspension in normal saline standardized to contain 100,000 organisms per milliliter. Both eyes of 16 guinea pigs were inoculated intracorneally with 0.25 ml. of the suspension. They were then divided into two groups, A and B, of eight animals each.

Treatment. Group A. The right eyes of the eight animals of this group received one drop of cortone suspension (5 mg. per cubic centimeter) hourly during the working day (8 a.m. to 5 p.m.). Simultaneously the left eyes were given one drop of the cortone suspending agent. At 4:30 on the first day of the experiment both eyes were injected subconjunctivally with 0.05 ml. cortone.

Group B. The right eyes of the eight animals of this group received one drop of terramycin and one drop of cortone suspension hourly. Simultaneously the left eyes were given only the terramycin solution. At 4:30 p.m. of the first day the

right eyes received 0.05 ml. of cortone, and at the same time all eight pigs were given 25 mg. terramycin orally.

Results. Same as in Experiment 1.

Summary, Experiment 3. Neither cortisone nor the suspending vehicle appeared to have any protective or deleterious effect on the course of the otherwise untreated corneal infections in Group A, which went on to loss of the eyes in all animals, nor to aid or damage the protective effect of terramycin in the Group B series.

Experiment 4. The Effect of Cortisone on Ulcers Produced Experimentally with Diplococcus pneumoniae, Type II

Materials and methods. A nineteen-hour culture on blood agar of a strain of type II pneumococcus isolated from a pneumococcic ulcer was used to prepare a suspension in normal saline standardized to contain 100,000 organisms per milliliter. Both eyes of 16 guinea pigs were inoculated intracorneally with 0.025 ml. of the suspension. They were then divided into two groups, A and B, of eight animals each.

Treatment. Same as in Experiments 1 and 3.

Results. Same as in Experiments 1 and 3.

Summary, Experiment 4. Neither cortisone nor the suspending vehicle appeared to have any protective or deleterious effect on the course of the pneumococcic corneal infections in this experiment; i.e., the eight animals of Group A treated with cortisone alone developed hopelessly necrotic eyes and those of Group B treated with cortisone and terramycin were completely cured.

Experiment 5. The Effect of Cortisone on Minimal Corneal Abscesses Produced Experimentally by Pneumococci

Materials and methods. A twenty-two-hour blood broth culture of pneumococci isolated from a corneal ulcer was used to prepare a suspension standardized to contain 150,000 bacteria per milliliter. Both eyes of four guinea pigs were injected intracorneally with 0.05 ml. of the suspension. This concentration had previously been found to be the lowest dilution capable of producing minimal infection. All eight corneas developed varying degrees of infection ranging from infiltration to an abscess in one case.

Treatment. The eye of each animal which developed the more pronounced infection was treated with cortisone suspension instilled into the conjunctival sac at half-hour intervals for 6.5 hours the first day and every hour the second day. The other eye of each animal was treated in the same manner with cortisone vehicle.

Results. At the end of 48 hours the cortisone-treated eyes continued to be slightly more involved than the vehicle-treated eyes. Six days after the inoculation all eyes but one vehicle-treated eye were clear.

Summary, Experiment 5. Cortisone did not appreciably affect the minimal lesions in this experiment.

Experiment 6. The Effect of Hydrocortisone on Minimal Corneal Ulcers Produced Experimentally with Pseudomonas aeruginosa

Materials and methods. A twenty-four-hour agar slant culture of a strain of Pseudomonas aeruginosa, previously determined to be of low virulence for the rabbit cornea, was used for the inoculation of both eyes of two rabbits. The inoculation was made by dipping the blade of a 5 mm. trephine into a thick suspension of the organism and making a one-quarter turn of the trephine on the pupillary area of the rabbit's cornea.

Treatment. The right eye of each rabbit was given a 0.2 ml. injection of ophthalmic hydrocortisone subconjunctivally at 12 o'clock; at the same time hydro-

cortisone ophthalmic ointment was instilled and the eye closed overnight.

Results. At the end of 48 hours ulceration had developed in the right hydrocortisone-treated eye of each rabbit and in one of the two untreated eyes. In the other untreated eye there was evidence of ulceration the following day but in this animal the ulcer was more severe in the treated eye than in the untreated eye until after six days; at this time the condition of the two eyes had become about equal.

Summary, Experiment 6. In this experiment hydrocortisone appeared to favor the development of infection.

Experiment 7. The Effect of Cortisone and Hydrocortisone on the Treatment of Minimal Corneal Ulcers Produced Experimentally with Pseudomonas aeruginosa

Materials and methods. The eight eyes of four rabbits were inoculated with Pseudomonas aeruginosa as in Experiment 6. Two of the rabbits developed bilateral ulcers during the first twenty-four hours and were treated as follows:

Treatment. Twenty-four hours after inoculation, when ulceration was in the incipient stage, the right eye of one rabbit was given 0.2 ml. of hydrocortisone ophthalmic suspension subconjunctivally near the limbus at 12 o'clock, and hydrocortisone ophthalmic ointment was instilled into the conjunctival sac three times daily. The right eye of the second rabbit was treated similarly with cortisone ophthalmic suspension and ointment. Both left eyes were left untreated.

Results. At the end of twenty-four hours of treatment the treated eyes of both rabbits showed decidedly less inflammatory reaction and less exudation than the untreated eyes, but the corneal lesions had progressed equally in all four eyes.

Summary, Experiment 7. In this experiment cortisone and hydrocortisone appeared to reduce the inflammatory phase of the ulceration without affecting the progress of the disease.

Experiment 8. A Further Experiment on the Effect of Cortisone and Hydrocortisone on the Treatment of Minimal Corneal Ulcers Produced Experimentally with Pseudomonas aeruginosa

Materials and methods. The fourteen eyes of seven rabbits were inoculated with an eighteen-hour culture of the same strain as that used in Experiment 7 and according to the same trephine technic. After twenty-four hours all the eyes showed infection which varied considerably in degree from eye to eye.

Treatment. The right eyes of the first three rabbits were treated with cortisone; a 0.1 cc. suspension was injected subconjunctivally daily and cortisone ointment was instilled into the conjunctival sac three times daily. The right eyes of the remaining four rabbits were treated similarly with hydrocortisone suspension and ointment. The left eyes in all seven rabbits were left untreated.

Results. At the end of twenty-four hours of treatment the infection in all eyes had progressed steadily except the infection of two cortisone-treated eyes which remained stationary. At the end of forty-eight hours the infections in two of the cortisone-treated eyes were definitely receding while in the control eyes of these rabbits the disease was progressing. In the third cortisone-treated animal a progressive infection was present in both the treated and the untreated eyes. In the four hydrocortisone-treated animals the infections were progressing in both treated and untreated eyes but the inflammatory reaction was definitely milder in the treated eyes. At the end of seven days there was

little difference between the treated and untreated eyes except in one hydrocortisone-treated eye in which there was definite ectasia of the cornea due to thinning although no perforation had taken place at the time the animals were sacrificed.

Summary, Experiment 8. In this series the variation in the severity of the ulcers produced precluded the drawing of any definite conclusions: the two eyes with the mildest ulcers received cortisone therapy while the eye with the severest ulcer was treated with hydrocortisone.

DISCUSSION

Ample clinical evidence is now available to indicate that topical cortisone and hydrocortisone have great value in controlling certain important ocular diseases arising from bacterial allergies. In phlyctenular keratoconjunctivitis it is now possible, by aborting the acute attacks of the disease, to prevent the often visually disastrous corneal complications. Phlyctenulosis is an excellent example of a disease in which the immune mechanisms of a primary remote tuberculous infection act unfavorably to the host and in which the inflammation-blocking effect of the hormones is of great clinical value. The mechanism of nongranulomatous uveitis is apparently similar and here again the protective action of the hormones seems unquestioned. It is probable that the marginal infiltrate or ulcer, so common in staphylococcic blepharoconjunctivitis of long duration, is also a manifestation of bacterial allergy, and here again the hormones are capable of aborting the lesion rapidly and of preventing scar formation. The marginal infiltrate and ulcer of rosacea, in the writer's opinion a manifestation of secondary staphylococcic infection, as well as typical rosacea keratitis, have reacted similarly.

Apart from these manifestations of bacterial hypersensitivity in which the causal bacteria are not in the tissue concerned but are either in a remote focus, as in the case of the tubercle bacillus in phlyctenular keratitis, or on the lid margins and conjunctiva, as in the marginal ulcer, no clear-cut clinical or experimental examples are available of any important beneficial effects of these hormones on ocular infections. Since the components of inflammation act to limit infection, interference with inflammatory phenomena can well result in a masking effect which is sometimes disastrous. This has certainly been the case in herpes simplex virus keratitis under both experimental and clinical conditions. On the other hand, inhibition of the inflammatory phenomena is not harmful when effective chemotherapy is used simultaneously. This was the case in the experimental corneal abscesses and ulcers produced by staphylococci and pneumococci.

It is of special interest that Irvine[19] has recently reported an instance of perforation of the cornea which developed in a cortisone-treated case of dendritic keratitis. An additional similarly treated case has been reported to us by Dr. Merrill Reeh, and one of us (P. T.) has seen in consultation a very severe perforating hypopyon ulcer which developed in the right eye of a woman with bilateral herpes corneae after topical cortisone therapy. No bacteria could be recovered from this ulcer but budding yeast forms, tentatively identified as Candida albicans (monilia) were found. Cortisone had been used until the ulcer was well established. Antibiotic therapy failed to influence its evolution. A fourth instance of perforation of the cornea in dendritic keratitis after cortisone therapy was reported to us by Dr. T. Frissel of Highland Hospital. His patient had been referred to the

hospital because of a ring ulcer. The ulcer progressed to perforation within twenty-four hours after hospital admission. No significant bacteria were demonstrable in cultures from the ulcer.

While this tendency of cortisone to cause perforation has not as yet been reported in other types of corneal ulcer, its tendency when used systemically to induce perforation of gastric and duodenal ulcers has received much attention in the literature. The exact mechanism of this effect has not been explained. In a letter to the *British Medical Journal*, Wybar expressed the opinion that cortisone might be contraindicated in deep corneal ulcers because of the experimental finding of delayed fibrous tissue reaction in corneal wounds.

Hypopyon ulcers due to pneumococci and other bacteria would seem to offer the best clinical means of evaluating the effect of cortisone owing to the ease with which they may be observed under the slit-lamp and corneal microscope. In the three pneumococcic cases reported in this paper, cortisone applied topically and alone was ineffective in preventing extension of the process during the first 24 hours but did not appear to influence healing when terramycin was used systemically and topically. The favorable results reported in 10 of 14 cases of hypopyon ulcer (causes not stated) by Duke-Elder and associates are difficult to evaluate in view of the experimental studies here reported.

It seems clear that great care should be exercised in the use of cortisone and hydrocortisone in central corneal ulcers and that a bacteriologic diagnosis should be made prior to employment of these hormones. If organisms for which successful chemotherapy or antibiotic therapy is available are present, the use of hormone therapy is justifiable. If organisms such as Pseudomonas aeruginosa are encountered, for which antibiotic therapy is less effective, hormone therapy should be delayed, at least until sensitivity tests show that susceptibility to an antibiotic exists.

On theoretic grounds cortisone would be expected to exert a favorable influence on those bacterial corneal infections fully susceptible to antibiotics by reducing vascularization and the production of granulation tissue and the resultant visual loss due to scar formation. In less susceptible infections cortisone would seem to be contraindicated in the initial stages, but in the late reparative stage some value might be gained. In only slightly susceptible or insusceptible infections, such as Pseudomonas infection, it would seem to be contraindicated since its interference with the normal tissue responses to infection would overbalance any favorable effect in limiting vascularization and granulation tissue formation. At the moment there is insufficient clinical evidence to prove or disprove these assumptions but the experimental evidence tends to be confirmatory.

A review of the literature and personal experience with experimental herpes simplex virus and experimental vaccinia virus keratitis indicate that the unfavorable effect of cortisone on viral infections is more pronounced following systemic therapy than following topical therapy. In view of the reports that ACTH is less likely to aggravate infection than cortisone, it should perhaps be used in preference to cortisone when systemic therapy is indicated.

SUMMARY AND CONCLUSIONS

1. The literature on cortisone as related to infections, and to corneal infections in particular, is reviewed. It can be concluded that while this hormone has no direct effect on microbial agents themselves, it does affect profoundly the body defenses and the body reactions to

injury. This effect is favorable in certain instances, as in the local manifestations of bacterial allergy, and unfavorable in other instances, as in the dissemination of tubercle bacilli from systemic foci. With respect to ocular infections, there is general agreement that cortisone and hydrocortisone, used topically, are effective in phlyctenulosis which is a manifestation of bacterial allergy, usually to a systemic tuberculous focus; but there is disagreement as to the effect of cortisone on hypopyon ulcers, both favorable and unfavorable results having been reported.

2. In a clinical study, cortisone and hydrocortisone administered topically were effective in the rapid control of all marginal ulcers in a series of 23 so-called catarrhal ulcers. Twenty of these were secondary to a staphylococcic blepharoconjunctivitis and 3 secondary to seborrheic blepharoconjunctivitis. Cortisone was ineffective in 2 cases of Mooren's ulcer, although it apparently effected a slight diminution of the inflammatory phase. Cortisone failed to influence 3 cases of central ulcers, all pneumococcic, which later responded to terramycin therapy. It also failed to influence a central ulcer of unknown cause which had not responded to sulfonamides or antibiotics. It influenced favorably each of 11 cases of phlyctenular keratitis, one believed to be secondary to staphylococcic blepharitis, the others to tuberculosis. It was effective in 2 cases of marginal ulcers in rosacea—ulcers believed to be due to secondary staphylococcic infection. It resulted in rapid healing of a marginal ulcer in a case of vernal catarrh with secondary Staphylococcus aureus infection. Cortisone and hydrocortisone had a masking effect only in early dendritic ulcers; in 2 of 13 cases placed on topical cortisone therapy, unusually severe deep corneal and uveal disease developed and progressed to complete loss of visual function. In late and trophic stages, however, the hormones appeared to be beneficial, both subjectively and objectively.

3. Cortisone was employed topically and systemically in the guinea pig in the treatment of experimental corneal ulcers due to Staphylococcus aureus, Diplococcus pneumoniae, and Pseudomonas aeruginosa. It had no protective effect in any instance. It did not appear to detract from or to enhance the effect of antibiotic therapy in a parallel series of experimental ulcers. Hydrocortisone employed by subconjunctival injection and in ointment form appeared to increase susceptibility to experimental Pseudomonas aeruginosa keratitis of the rabbit.

4. It is concluded that cortisone and hydrocortisone are potent hormones capable of modifying profoundly the tissue reaction to infection. When this tissue reaction is so violent as to be harmful, the anti-inflammatory action of cortisone can be useful, but by interfering with tissue defenses the cortisone may mask the progress of the infecting agent. Cortisone should therefore be used with caution except in those diseases in which effective chemotherapy is available.

REFERENCES

1. Abernathy, Robert: The effect of cortisone on experimental brucellosis, J. Clin. Investigation, 30:626 (June) 1951.
2. Arruga, H.: La cortisone en ophthalmologia, Arch. Soc. oftal. hispano-am., 11:585-603 (June) 1951.
3. Barrios, R. R., and Barriere, R. V.: La cortisone en oftalmologia, Arch. Soc. oftal. hispano-am., 11:276-288, 1951.
4. Bjørneboe, Mogens, Fischel, Edward E., and Stoerck, Herbert C.: The effect of cortisone and adrenocorticotrophic hormone on the concentration of circulating antibody, J. Exper. Med., 93:37-48 (Jan. 1) 1951.

5. Bloch, Robert G., Vennesland, Kirsten, and Gurney, Clifford: The effect of cortisone on tuberculosis in the guinea pig, J. Lab. & Clin. Med., 38:133-147 (July) 1951.

6. Boling, L., Newkirk, J., Baxter, P., Partridge, J., Margen, S., and Kinsell, L. W.: The use of ACTH in the management of generalized peritonitis: a preliminary report, J. Clin. Endocrinol., 12:184-190 (Feb.) 1952.

7. Bruckner, R.: Zur lokalen Anwendung von Cortisone bei Augenerkrankungen, Ophthalmologica, 121:275-286, 1951.

8. Collins, Conrad B., Davidson, Vanda A., and Mathews, Neilson M.: Use of cortisone in pelvic cellulitis; preliminary report, New Orleans M. & S. J., 104:389-394 (April) 1952.

9. Desvignes, P., Fontaine, and Morault, Y.: Premiers essais de traitement des iridocyclites par la cortisone à l'Hotel-Dieu, Bull. Soc. Ophtal. France, 9:776-784, 1950.

10. Duke-Elder, Stewart, Duthie, O. M., Foster, J., Law, F. W., Lister, A., Reid, Mckie, Scott, G. I., Zorab, E. C., and Goldsmith, A. J. B.: A series of cases treated locally by cortisone; a preliminary report to the medical research council by the panel on the ophthalmological applications of cortisone and ACTH, Brit. J. Ophth., 35:672-694 (Nov.) 1951.

11. Fine, Max, and Goodwin, Rufus C.: Evaluation of local cortisone therapy in ophthalmology, Arch. Ophth., 47:787-797 (June) 1952.

12. Fitzgerald, J. Robert, Bellows, John G., Donegan, Justin M., Gamble, Richard C., Krause, Arlington C., Mann, William A., Pearlman, Maurice D., and Zekman, Theodore N.: Early clinical results of ACTH and cortisone treatment of ocular disease, Arch. Ophth., 45:320-333 (March) 1951.

13. Franceschetti, A., Maeder, G., and Bourquin, J. B.: La cortisone en ophtalmologie (Observations experimentales et cliniques), Ann. d'ocul., 185:35-64 (Jan.) 1952.

14. Germuth, F. G., Jr., Ottinger, B., and Oyama, J.: The influence of cortisone on the evolution of acute infection and the development of immunity, Bull. Johns Hopkins Hosp., 91:22 (July) 1952.

15. Glaser, Robert J., Berry, John W., Loeb, Lenore H., and Wood, W. Barry, Jr.: The effect of cortisone in streptococcal lymphadenitis and pneumonia, J. Lab. & Clin. Med., 38:363-373 (Sept.) 1951.

16. Hallett, Joseph W., Leopold, Irving H., Vogel, Adolph W., Cannon, Edmond W., and Steinmetz, Charles G.: Treatment of experimental herpes simplex keratitis in the rabbit, Arch. Ophth., 46:33-38 (July) 1951.

17. Hurtig, A.: Use of cortisone and antibiotics in resistant pelvic infections, Postgrad. Med., 11:196-201 (Feb.) 1952.

18. Ikui, H.: Topical use of cortisone in ocular inflammation, Rinsho-Ganka-Iho., 45: 532-534 (Oct.); 595-596 (Nov.); 657-660 (Dec.) 1951.

19. Irvine, S. Rodman: ACTH and cortisone in ophthalmology, Ann. West. Med & Surg., 6:438-440 (July) 1952.

20. Irvine, S. Rodman, and Irvine, Mary Dailey: The effect of cortisone on the primary and secondary aqueous and on corneal vascularization in rabbits, Bull. Johns Hopkins Hosp., 89:288-302 (Oct.) 1951.

21. James, William M., Power, John L., and Ripple, Paul H.: Experimental and clinical observations of the effects of cortisone in corneal lesions, Am. J. Ophth., 35:1298-1304 (Sept.) 1952.

22. Kilbourne, E. D., and Horsfall, F. L.: Lethal infection with coxsackie virus of adult mice given cortisone, Proc. Soc. Exper. Biol. & Med., 77:135-138 (May) 1951.

23. Kimura, S. J., Thygeson, P., and Geller, H. O.: Observations on the effect of adrenal steroids on vaccinia virus. I. The effect of cortisone in experimental vaccinia virus keratoconjunctivitis of the rabbit, Am. J. Ophth. (In press.)

24. Kligman, Albert M., Baldridge, G. Douglas, Rebell, Gerbert, and Pillsbury, Donald M.: The effect of cortisone on the pathologic responses of guinea pigs infected cutaneously with fungi, viruses, and bacteria, J. Lab. & Clin. Med., 37: 615-620 (April) 1951.

25. Lavery, F. S., Werner, L. E., O'Donoghue, D., Guinan, P. M., and Macdougald, J.: Use of cortisone in diseases of the eye, Brit. M. J., 35:1285-1289 (June) 1951.

26. Leopold, Irving H., Purnell, James E., Cannon, Edmond J., Steinmetz, Charles G. and McDonald, P. Robb: Cortisone in ocular disease, Am. J. Ophth., 34:361-371 (March) 1951.

27. Lepri, G.: Recherche sulla funzion alita cortico-surrenalica nella cheratete rosacea e trattamento terapeutica con ormone adrenocorticotropica (ACTH) ed acido ascorbico, Arch. ottal., 54:109-134, 1950.

28. Lepri, Giuseppe: Studies on cortisone in ophthalmology. I. The influence of cortisone on the in-vitro effect of antibiotics on microörganisms capable of causing ocular infections. II. The action of cortisone on experimental pneumococcic infection of the cornea and its influence on the activity of several antibiotics, Am. J. Ophth., 35:935-946 (July) 1952.

29. Lerner, M.: Dendritic ulcer treated with cortisone, Brit. J. Ophth., 35:435-436 (July) 1951.

30. Marcus, Stanley, and Donaldson, David M.: Suppression of Shwartzman phenomenon by adrenocorticotropic hormone and cortisone; quantitative aspects, J. Immunol., 69:101-108 (July) 1952.

31. McLean, John M., Gordon, Dan M., and Koteen, Herbert: Clinical experiences with ACTH and cortisone in ocular diseases, Tr. Am. Acad. Ophth., 55:565-572 (May-June) 1951.

32. Michael, M., Jr., Cummings, M. M., and Bloom, W. L.: Course of experimental tuberculosis in the albino rat as influenced by cortisone, Proc. Soc. Exper. Biol. & Med., 75:613-616 (Nov.) 1950.

33. Mogabgab, William J., and Thomas, Lewis: The effects of cortisone on bacterial infection, J. Lab. & Clin. Med., 39: 271-289 (Feb.) 1952.

34. Moser, R.: Cortison bei Keratitis disciformis und Herpes simplex corneae, Schweiz. med. Wchnschr., 81:945-946 (Sept.) 1951.

35. Mosher, Henry Adams: Local use of cortisone in ophthalmic diseases, Arch. Ophth., 45:317-319 (March) 1951.

36. Olson, J. A., Steffensen, E. H., Smith, R. W., Margulis, R. R., and Whitney, E. L.: Use of adrenocorticotropic hormone and cortisone in ocular disease, Arch. Ophth., 45:274-300 (March) 1951.

37. Ormsby, H. L., Dempster, G., and van Rooyen, C. E.: Effect of cortisone on experimentally induced herpetic keratitis of the rabbit, Am. J. Ophth., 34:1687-1692 (Dec.) 1951.

38. Ormsby, H. L., Shirley, S. Y., Aikenhead, J. S., and Murray, R. G.: ACTH and cortisone therapy in eye disease, Canad. M.A.J., 66:62-66 (Jan.) 1952.

39. Plotz, Charles M., Howes, Edward L., Blunt, J. Wallace, Meyer, Karl, and Ragan, Charles: Action of cortisone on mesenchymal tissue, Arch. Dermat. & Syph., 61:919-921 (June) 1950.

40. Purnell, James E., and Leopold, Irving H.: Cortisone in ocular disease; further studies, Am. J. Ophth., 35:663-670 (May) 1952.

41. Raiford, M.: The use of cortisone and ACTH in ophthalmology, Guildcraft, April, 1951, p. 15.

42. Radaelli P., Cavallero C., Borasi M., Sala G., and Amira A.: Infezione sperimentale da Coccidiodes immitis e steroidi cortisurrenali, Mycopath. et Mycol. Appl. 6:7-14, 1951.

43. Reveno, William S., Rosenbaum, Herbert, and Buell, John H.: Short-term use of corticotropin as adjunctive therapy, J.A.M.A., 149:1308-1309 (Aug. 2) 1952.

44. Robinson, H. J.: Effects of cortisone on intradermal pneumococcal infections in rabbits, Proc. Soc. Exper. Biol. & Med., 10:372, 1951.

45. Rouher, F.: Les instillations de cortisone dans quelques affections oculaires, Bull. Soc. d'opht. Paris, Feb. 1951, pp. 138-140.

46. Scheie, Harold G., Tyner, George S., Buesseler, John A., and Alfano, Joseph E.: Adrenocorticotropic hormone (ACTH) and cortisone in ophthalmology; report of cases, Arch. Ophth., 45:301-316 (March) 1951.

47. Shwartzman, Gregory: Poliomyelitis infection in cortisone-treated hamsters induced by the intraperitoneal route, Proc. Soc. Exper. Biol. & Med., 79:573-576, 1952.

48. Shwartzman, G., and Fisher, A.: Alteration of experimental poliomyelitis infection in the Syrian hamster with the aid of cortisone, J. Exper. Med., 95:347-362 (April) 1952.

49. Steffensen, E. H., Ivy, H. G., and Nagle, F. O.: Topical Compound F in the treatment of anterior segment eye disease, Am. J. Ophth., 35:933-934 (July) 1952.

50. Steffensen, E. H., Wishbow, A. J., Nagle, F. O., Smith, R. W., and Whitney, E. L.: Topical cortisone in the treatment of anterior-segment eye disease, Am. J. Ophth., 34:345-356 (March) 1951

51. Stoerck, H. C.: Inhibition of tuberculin reaction by cortisone in vaccinated guinea pigs, Proc. Soc. Exper. Biol. & Med., 9:345, 1950.

52. Teilum, G., Enghaek, H. C., and Simonsen, M.: Effects of cortisone on plasma cells and reticulo-endothelial system in hyperimmunized rabbits, Acta endocrinol., 5:181-193, 1950.

53. Thorpe, H. E.: ACTH and cortisone in ocular trauma and in eye surgery; a preliminary report, Proc. Second Clinical ACTH Conf. Blakiston Co., 1951, pp. 340-361. Also personal communication to the authors.

54. Thygeson, Phillips, and Fritz, Milo H.: Cortisone in the treatment of phlyctenular keratoconjunctivitis, Am. J. Ophth., 34: 357-360 (March) 1951.

55. Thygeson, Phillips, Geller, Hildegard O., and Schwartz, Ariah: Effect of cortisone on experimental herpes-simplex keratitis of the rabbit, Am. J. Ophth., 34:885-888 (June) 1951.

56. Thygeson, P., Hogan, M. J., and Kimura, S. J.: This communication.

57. Turner, T. B., and Hollander, D. H.: Cortisone in experimental syphilis; a preliminary note, Bull. Johns Hopkins Hosp., 87:505-509 (Nov.) 1950.

58. Von Sallmann, L.: Personal communication to the authors.

59. Woods, Alan C.: Use and limitations of ACTH and cortisone in ophthalmology, Brit. J. Ophth., 36:401-431 (Aug.) 1952.

60. Woods, Alan C., and Wood, Ronald M.: The effect of cortisone and ACTH on ocular inflammation secondary to the injection of irritant substances, Bull. Johns Hopkins Hosp., 90:134-148 (Feb.) 1952.

61. ———: Studies in experimental ocular tuberculosis. XVI. Effect of corticotropin, cortisone and Compound F on development of experimental ocular tuberculosis in immune-allergic and normal rabbit, Arch. Ophth., 47:477-512 (April) 1952.

62. Woodward, T. E., Hall, H. E., Dias-Rivera, R., Hightower, J. A., Martinez, E., and Parker, R. T.: Treatment of typhoid fever. II. Control of clinical manifestations with cortisone, Ann. Int. Med., 34:10-19 (Jan.) 1951.

DISCUSSION

JAMES H. ALLEN, M.D., New Orleans, La.: I wish to commend Dr. Thygeson on his paper. It is an excellently designed and executed experimental and clinical study and evaluation of cortisone. I believe it is unfortunate he did not have time to show you the designs of his experiments. They were very well controlled.

For some time we have been using cortisone clinically, but from the beginning, very fortunately, I believe, we were skeptical about the use of cortisone in the presence of active infectious processes. For that reason we cannot substantiate some of Dr. Thygeson's findings, although we can emphasize the didactic and theoretic basis for the poor results, or the lack of results, that he obtained. We can also verify the good effects of cortisone therapy in some of the noninfectious lesions of the conjunctiva and cornea, particularly in vernal conjunctivitis of the limbus variety with the marginal corneal lesions. The lesion seems to melt in twenty-four to forty-eight hours under adequate cortisone therapy.

In certain cases that were questionably allergic, such as interstitial keratitis, we have taken an experimental attitude with regard to the use of cortisone. Instead of instituting cortisone immediately in those cases, we insisted that they be on active and intensive antiluetic therapy for a minimum period of twenty-four hours before cortisone therapy was instituted. In five of those cases we have had what we consider to be excellent results and more rapid healing than we would have expected otherwise, with unusual clearing of the cornea.

In a number of other noninfectious marginal lesions of the cornea, we have been very happy with the use of cortisone. We have been impressed with the fact that perhaps the drug may be of some benefit in the course of leprotic lesions of the cornea, providing the patient is under intensive antileprotic treatment at the time.

On the other hand, we believe that there are some very definite contraindications to the use of cortisone therapy from clinical observations that we made. Although we have not used the drug ourselves in all of these cases, we have seen cases in consultation which have been referred to us after cortisone had been used. Three cases of dendritic keratitis came to our attention under that category. In 2 cases the lesion had been prolonged over a period of three months, with the result that when finally the lesion was healed there was intensive scarring of the superficial cornea. In the first case the cornea became necrotic. An extensive slough occurred in the center of the cornea, and there was some question as to whether the globe might rupture for some time before the cornea finally healed. In other words, we believe there was an exacerbation

or an increase in the virulence of the infection as a result of the cortisone therapy.

In 2 cases of ulcers of the central portion of the cornea that we saw in consultation we were impressed with the fact that the marginal infiltration of the cornea was not present, as you would characteristically expect. In one case, however, the central lesion had progressed to necrosis of the cornea and the globe was lost. In the second case an extensive corneal ulceration occurred and the visual result was less than 20/200 when the lesion healed; this in spite of the fact that both patients were on intensive antibacterial therapy which should have controlled the infection.

For this reason I should like to re-emphasize Dr. Thygeson's summary and conclusions that one should exert extreme care in the use of cortisone in infectious diseases of the conjunctiva even though active antibacterial agents may be used. I would suggest that extreme care be used in adding cortisone to the therapy of those individuals, and I should like to add one word of caution in that regard. We cannot assume that because most organisms of a given type respond to a particular drug, all organisms will be killed or inhibited by that particular antibacterial agent. There is an increasing number of strains of all types of organisms showing resistance to the various antibacterial agents, and where there is a question of the effectiveness of the antibacterial agent, cortisone should be held off or not used. If there is the need of an additional therapeutic agent, we feel that perhaps a safer form of therapy would be the addition of foreign protein therapy. The reason is that foreign protein therapy does stimulate hormones within the body, but in addition to that it stimulates very actively the entire defense mechanism of the body.

IRVING H. LEOPOLD, M.D., Philadelphia, Pa.: Drs. Thygeson, Hogan and Kimura in typically thorough fashion reviewed the role of cortisone in nonbacterial and bacterial keratitis. They have concluded from their study of the literature and from their own personal experimental and clinical observations that these hormones are of definite value in certain forms of nonbacterial keratitis, and in bacterial keratitis only when there is an associated marked hypersensitivity. Specifically they have found beneficial effects from these hormones in marginal ulcers, in the ulcers associated with acne rosacea and in phlyctenular keratitis. They were unable to demonstrate any favorable influence of these hormones on purely bacterial keratitis. They have, therefore, suggested that these hormones not be used in bacterial keratitis unless there is a marked and violent inflammatory reaction and unless one can employ a specific antibiotic along with the hormonal therapy.

There can be no argument with these conclusions. Actually they are confirmation of the earlier warnings of Swan in 1950; Armstrong and Irons in 1951; of Swedberg and his co-workers; of Lepri and Williams; of Woods and many others, who have stressed that the nonspecific blocking action on inflammation of these hormones associated with infection may be undesirable. Woods also reviewed the literature a short while ago, (Brit. J. Ophth., Aug. 1952) and from his own personal clinical and experimental experience particularly has concluded that these agents should not be employed to interfere with or inhibit the inflammatory reaction associated with infection unless one can depend on the natural resistance of the host to overcome the infection or on a specific chemotherapeutic or antibiotic agent in addition to the hormone.

There are other factors that should be considered if one is going to employ an antibiotic along with these hormones. There is the factor of blood aqueous permeability and of capillary permeability. You are all aware that agents such as streptomycin and others penetrate poorly when administered systemically, but when the eye is inflamed these antibiotics penetrate into the infected tissue. It has been shown that cortisone will reduce the permeability of the inflamed eye, and if cortisone will reduce the permeability of the inflamed eye it might, therefore, interfere with the penetration of these antibiotics into the inflamed eye at just the time when we desire to have a higher concentration of these antibiotics in the tissues.

Studies were, therefore, undertaken at the Wills Hospital to determine the influence of these hormones on the blood aqueous behavior in the presence of inflammation. The antibiotic chosen for study was streptomycin, which penetrates extremely poorly into the normal eye, actually giving levels of from zero to 1 unit in the normal tissue. Benzyl alcohol has to be removed from the hormonal preparation. It is a preservative in cortisone and hydrocortisone preparations and will give a bacteriostatic level in itself. If it is removed, the preparation has very little bactericidal effect.

Briefly, you can see that the inflamed eyes which have been treated with local cortisone or hydrocortisone show very little difference in the penetration of systemically administered streptomycin into the aqueous or vitreous humor. When cortisone is given systemically, the effect on the penetration of streptomycin into the inflamed tissue is not significant. It appears, then, that these hormones will not interfere with the permeability of the inflamed eye for antibiotics like streptomycin when the inflammation is moderate or severe.

Drs. Thygeson, Hogan and Kimura have shown that cortisone and hydrocortisone do not interfere with the action of terramycin. This again is confirmatory of the observations of others; namely, the work of Mogabgab and Lewis, who demonstrated that the cortisone did not interfere with the action of penicillin against streptococcic infection; of Glaser, who showed that penicillin could be used in the presence of cortisone against streptococcic infection; of Svedberg and his co-workers; Lepri and Williams; Woods and others, who have employed streptomycin or streptomycin and para-aminosalicylic acid along with these hormones in the treatment of tuberculosis without interference; and of Smadel, who has shown that chloramphenicol can be used with cortisone effectively against typhoid fever.

There are a few discrepancies between the findings of Thygeson, Hogan and Kimura and the results of others as reported in the literature.

For example, Smith and Opsahl in 1951 reported their results in infections with organisms containing hyaluronidase and with those free of hyaluronidase. They were able to show that cortisone had a favorable effect on experimental infection with an organism containing hyaluronidase, but when the organism was free of hyaluronidase, cortisone had no influence on the course of the infection. Drs. Thygeson, Hogan and Kimura employed both types of organisms in their experiments and no difference could be distinguished between those containing hyaluronidase and those free from hyaluronidase.

There is also room for interesting speculation concerning the wide difference in their results in the therapy of hypopyon keratitis and the many clinical reports of benefit in this condition. There is the report of Hart and Mather made in 1951 that by using controlled experimental hypopyon lesions they were able to show more rapid clearing of the eyes which were treated with cortisone.

Encouraging news is the slight, but favorable, report on the cases of Mooren's ulcers. This may be somewhat helpful as to the possible etiology of this condition and certainly gives us some hope that there may be a form of therapy for this very serious and to date unsatisfactorily treated disease.

Drs. Thygeson, Hogan and Kimura have, by virtue of their careful study, given us considerable help in identifying the limitations and proper position of these hormones in ophthalmic therapeutics.

DR. THYGESON: I should like to close the discussion by thanking Dr. Allen and Dr. Leopold for their excellent additions to the paper. Dr. Allen has had a unique opportunity to study leprotic keratitis in relation to treatment with the sulfones. I was particularly interested in Dr. Leopold's comments on permeability as related to antibiotics and cortisone, and we shall keep these in mind in future work. One possible reason for our failure to show differences in cortisone effect between organisms producing hyaluronidase and those that do not is the fact that experimental ulcers tend to be fulminating in nature. We attempted with some success in our last two experiments to produce minimal lesions with Pseudomonas aeruginosa and we expect to apply this technic to other organisms.

ACCIDENTAL SURGICAL INJURY TO THE LENS

LEONARD CHRISTENSEN, M.D.
and
EDWARD MCLEAN, M.D.
BY INVITATION
PORTLAND, ORE.

DURING the past two and a half years, a survey of the causes of blindness has been conducted in the state of Oregon. Emphasis has been focused on all circumstances leading to blindness rather than on a mere accumulation of clinical and pathological diagnoses. From this approach it has become apparent that loss of vision often results from factors other than the disease alone and that statistics based solely on clinical diagnoses are not wholly valid. For example, microscopic analyses of globes enucleated for intractable glaucoma indicated that faulty surgery often contributed heavily to the final result. Where glaucoma was the primary disease, enucleation occasionally followed because of, rather than in spite of, some decompression operation. Of the various types of technical accidents occurring in glaucoma surgery, our attention was especially directed toward the occurrence of injury to the lens capsule for the following reasons. First, the incidence of this complication was much greater than might be expected and almost without exception it had escaped clinical detection. Second, this accident was found to have a far more disastrous effect on the eventual course of the eye than simple opacification of the lens or blockage of a filtering bleb. Finally, failure to recognize its occurrence or the full significance of its long-range effect when recognized prevented adequate attempts to salvage the eyes. This paper is presented with the hope that there will be an increased awareness of the high incidence of this surgical accident and that greater effort will be made toward early recognition and better understanding of its full significance.

Of 47 enucleated eyes with a history or histological evidence of glaucoma surgery, 17 had perforation of the lens capsule. The surgical procedures included iridectomies, paracenteses and filtering operations of various types. Despite great variability in the duration of the glaucoma and postoperative course, these globes showed a uniform pattern of reaction to the injury. In the region of the surgical wound there was usually an abundance of granulation tissue which incorporated the iris and capsule of the lens and mechanically blocked the scleral opening (figs. 1 and 2). The amount and state of organization of this granulation tissue varied with time, degree of injury and tissue reactivity to lens protein. The dehiscence in the lens capsule was usually found in close approximation to the wound of entry and this capsule was often fragmented. Anterior and posterior synechiae generally were present. Within the lens metaplasia of the epithelium occasionally was noted from which a variable amount of fibrous connective tissue seemed to have originated. This connective tissue was often in direct continuity with fibroblastic tissue in the anterior chamber. Subcapsular concentrations of all types of inflammatory cells were common (fig. 3).

From the Department of Ophthalmology, University of Oregon Medical School.
Part of a study conducted under a grant from the Kellogg Foundation.

Presented at the Fifty-Seventh Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 12-17, 1952, Chicago, Ill.

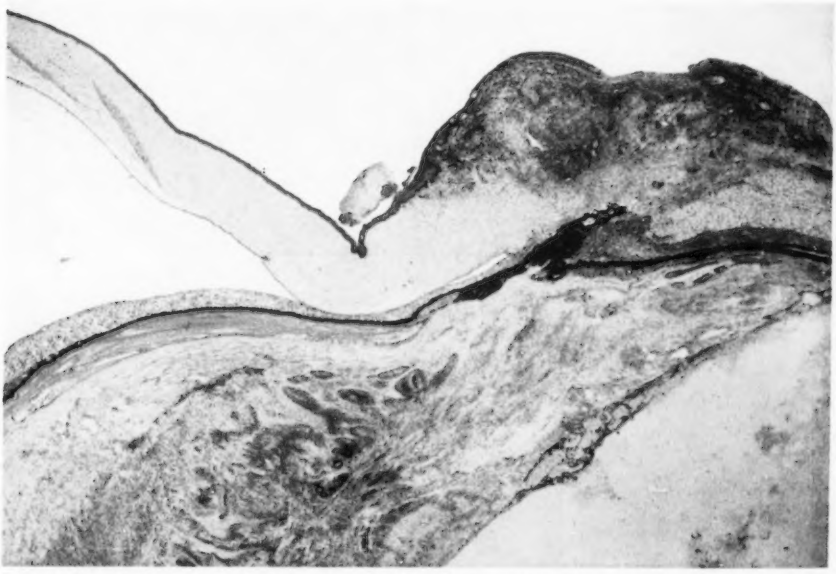


FIG. 1—Note the extensive granulation tissue in the region of the surgical wound. The lens is almost completely absorbed and the retrolental mass is detached retina.

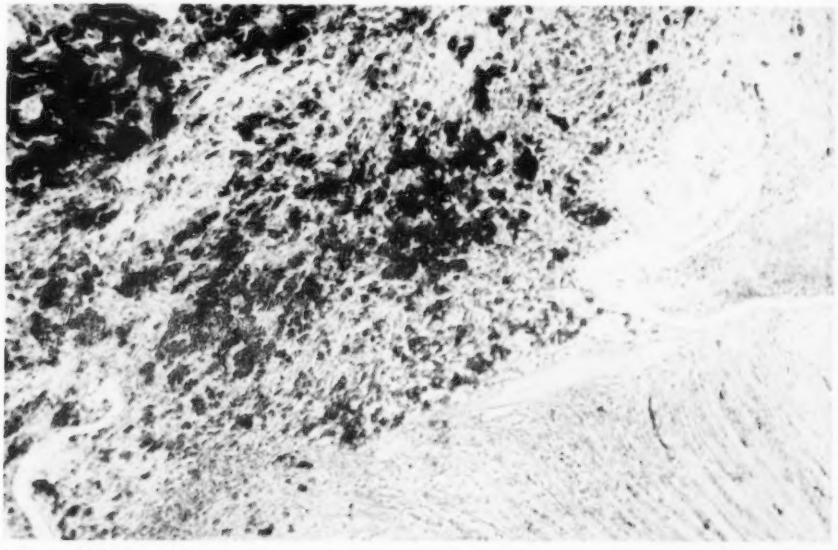


FIG. 2—High power showing lens capsule incarcerated in the fibrous connective tissue adjacent to the surgical wound.

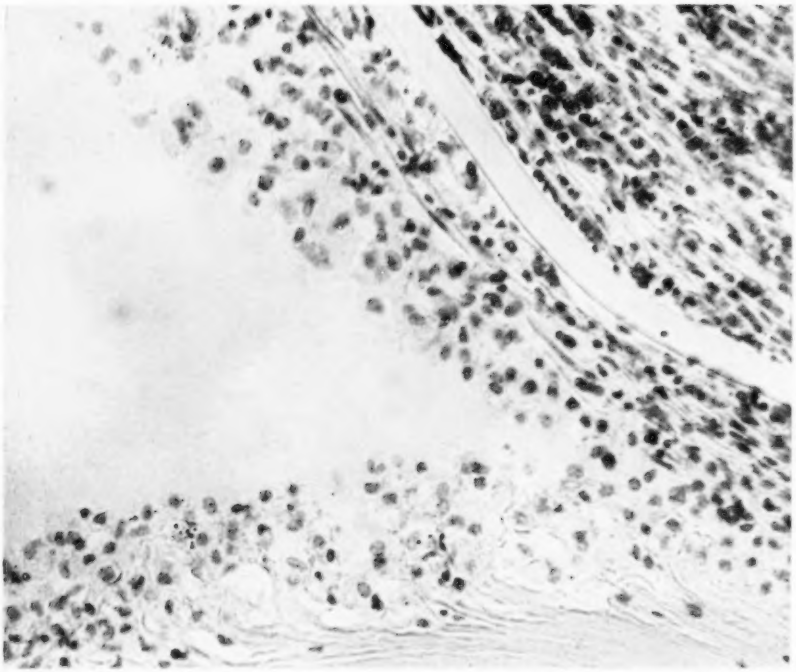


FIG. 3—High power showing subcapsular accumulation of inflammatory cells. The majority of these cells are macrophages.

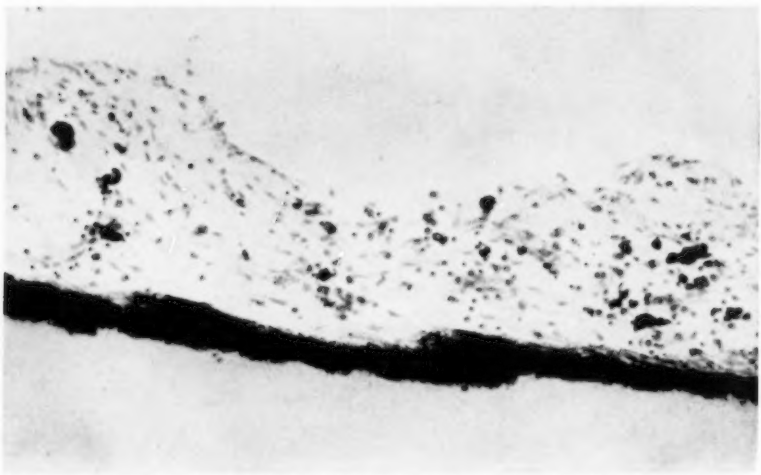


FIG. 4—Section of iris showing minimal infiltration by inflammatory cells. The stroma is atrophic.

FIG. 5—Note the moderately intense inflammatory and connective tissue reaction in the region of the iris and ruptured lens. The anterior chamber is filled with protein rich fluid.

Despite a long postoperative time interval in some cases, there was almost always an inflammatory reaction of some degree in the uveal tract, particularly in the iris. Furthermore, there usually was evidence of severe tissue reaction which could be attributed only to the inflammation. This was characterized by a proliferative fibroplasia of various stages occurring within the uvea, lens or scleral wound. For our purposes, this inflammatory reaction was classified into three categories according to intensity. The minimal reaction consisted of an infiltration within the stroma of the iris and ciliary body by plasma cells and lymphocytes (fig. 4). In this group few cells were present in the anterior chamber and there was no histological evidence of alteration of the aqueous. Occasionally, giant cells were seen in the lens or near the wound of entry. Clinically, the inflammation in these cases would have been difficult to recognize and the postoperative opacity of the lens might well have been attributed to other causes. The second group had the same type of cellular reaction but of much greater intensity (fig. 5). Cells were present in the anterior chamber, chamber angle and on the corneal endothelium. In addition there was frequently an acellular pink-staining substance within the anterior chamber representing protein rich fluid. In these globes aggregations of polymorphonuclear cells were occasionally found within the lens matrix. This group could

FIG. 6.—Gonioscopic view through the iris defect showing incarceration of the lens capsule and a dense nuclear opacity.

have been clinically recognized because of the floaters, visible beam and occasional keratic precipitates. The third group had a frankly purulent process. The violent polymorphonuclear reaction simulated in every respect an anaphylactoid reaction.

Failure of early clinical recognition and adequate therapy were undoubtedly important factors leading to the eventual loss of these eyes. Therefore, a study of patients who had had glaucoma surgery was made in an attempt to find a characteristic clinical pattern which might indicate lens injury. Sixteen patients who demonstrated or gave a history of rapidly developing postoperative cataract were studied. Seven showed absolute evidence of capsular injury. This consisted of a dehiscence in the capsule, visible gonioscopically. The postoperative period in this group ranged from several weeks to four years. In one of the seven seen soon after discharge from the hospital, a large break in the lens was noted just anterior to the equator. An opacity began early in this area and spread along the posterior subcapsular plane. Within three months, the entire lens was opaque. In this patient the eye became irritable, occasional floaters were seen in the aqueous and a beam was constantly visible. In five, there was a reduction in the depth of the anterior chamber and in three, almost complete loss. In one patient the glaucoma surgery was followed by an active uveitis. This led to the clinical diagnosis of primary uveitis with secondary glaucoma. The postoperative course was stormy and gonioscopy two years later showed dehiscence of the capsule and rupture of the zonular fibers. The cataractous lens was displaced toward the wound and the capsule incarcerated within the wound (fig. 6). In only one case did the lens opacity remain limited to the site of injury with no clinical evidence of uveitis.

Although these 7 cases represent a small number, a common postoperative pattern again was shown. In each instance where the lens injury was demonstrable, development of opacity was rapid and usually complete within three months. With one exception, some degree of uveitis was present and was demonstrated by a few floating cells, faintly visible beam and posterior synechiae. The most salient feature of this inflammatory reaction was its persistence over a prolonged period of time. The observation of postoperative shallowing of the anterior chamber seemed to have diagnostic significance. In addition to the filtering effect, it could occur because of swelling of the lens, posterior synechiae and anterior displacement of the lens iris diaphragm. The dehiscence in the capsule occurred adjacent to the wound of entry; however, failure to find a defect in the capsule did not rule out such an injury. In order to be visualized,

FIG. 7—Gonioscopic view of the eye with aniridia and congenital cataract. Note the incarceration of lens capsule and limitation of acquired opacity to the area of trauma.

the size of the dehiscence had to be large. This was observed in three patients with traumatic perforations of the cornea and lens. Even knowing the exact tract of the perforating fragments, we were unable to see the point of capsular entry. Therefore, we concluded that even with gonioscopy only the larger defects are visible.

Cataract following intraocular surgery has been recognized since von Graefe first introduced an iridectomy into the treatment of glaucoma.

The reported incidence of lens opacity following surgery has been variable and undoubtedly varies with the skill of the operator and type of surgery as well as the alteration of the unknown metabolic processes that maintain transparency.[2,3,8,9,12,17-21,22,23,24,26] From the literature it seems that approximately 20 to 30 per cent of ocular filtering procedures are followed by lens opacity within a few years. The percentage of these opacities attributed to mechanical injury of the lens is generally low and has ranged from one statement that it never occurs to an upper extreme of 10 per cent.[1,4,6,7,11,14,15,17,18,21,22,24] On the other hand, Verhoeff stated there was injury to the lens capsule in the majority of eyes he had examined histologically following unsuccessful trephination.[25]

Explanations for the lens opacity following glaucoma surgery have been numerous, the most common being prolonged hypotony, simple maturation of pre-existing senile changes and mechanical injury including rupture of the capsule. Most studies have been based on clinical observations without the aid of gonioscopy and usually the conclusions drawn were by inference only. Since hypotony frequently follows successful surgery and because it represents an alteration of ocular physiology, it seems a reasonable explanation for at least some postoperative cataracts.[5,18] However, from the present studies we feel this concept has been overemphasized.

The most significant common finding in this study has been the presence of a persistent inflammatory reaction in the uveal tract. This inflammation can be attributed to the ruptured lens. Previous reports by the Irvines and others substantiate this observation.[10,13,16] To appreciate fully the significance of this complication requires a fundamental understanding of inflammation. In brief, inflammation may be defined as reaction to injury and with it goes loss of function. In addition to a complex vascular and lymphatic reaction there is a local cellular infiltration and eventual cellular death. This is associated with or followed by an equally complex infiltration of the elements of repair, specifically neovascular and fibrous connective tissue.

Consider this in a glaucomatous eye. First, there is lens opacity and mechanical blockage of a filtering bleb (fig. 8).

FIG. 8.—Low power showing rupture of the capsule and displacement of the lens toward the wound. Note the intense reaction in the iris and around the area of the scleral defect.

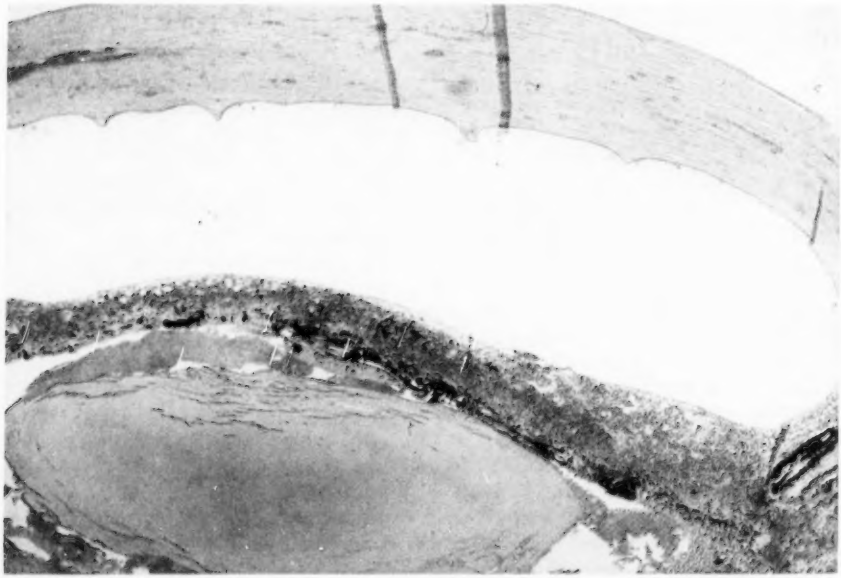


FIG. 9.—Low power showing the destructive effect of both the inflammatory reaction and elements of repair.

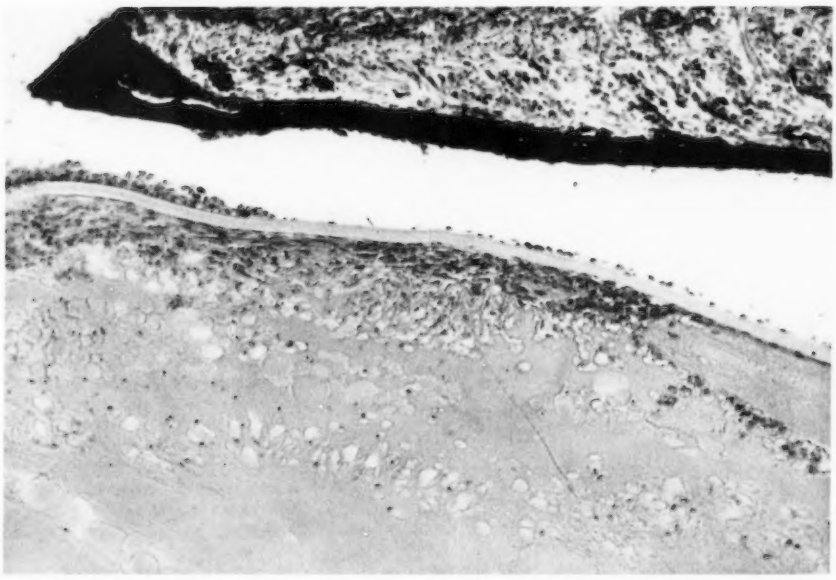


FIG. 10.—In this section there is metaplasia of the lens epithelium as shown by subcapsular formation of fibrous connective tissue.

More important, the rupture of the capsule sets off a chain of tissue reaction superimposed upon a pre-existing pathological state. This consists of a local and uveal inflammation from constant exposure to proteolytic products of a decomposing lens. The nature and intensity of this reaction are determined by the rate of cortical lysis, tissue sensitivity and tissue reactivity. Necrosis may be extensive and repair limited or there may be variation of the two. When the inflammation is overwhelming, necrosis predominates and there is dissolution of intraocular structures and profound impairment of normal metabolic processes. This may be followed by immediate loss of the globe or irreversible loss of function. When the reparative phase predominates, there may be replacement of iris stroma by fibrous connective tissue, closure of the chamber angle and posterior synechiae. This proliferation of connective tissue within the globe further embarrasses a previously altered metabolic state. Therefore, in an eye, unlike other organs, both the destructive and reparative phases can be equally disastrous (fig. 9).

This brings up the subject of treatment. In some cases if the lens defect is sufficiently small and no hypersensitivity exists, the uveitis may be aborted by closure of the defect in the lens. This can be accomplished in part by metaplasia of the lens epithelium. There is evidence that this takes place (fig. 10). Under these circumstances, no immediate surgical procedures would be indicated. However, where the uveitis is moderately intense and persistent, a more radical approach might be considered. Despite the well-established surgical principle to avoid lens extraction in the face of an active uveitis, it would seem the most advisable procedure under the above circumstances.

CONCLUSIONS

In this clinical and pathological study, injury to the lens capsule during glaucoma surgery was found to be higher than is generally accepted. Furthermore, evidence was found indicating this injury induces an inflammatory reaction within the uvea that may lead to the eventual loss of an eye.

Gonioscopy is a useful aid in diagnosis of the injury but minor defects can easily escape detection.

Early extraction of the injured lens may be the treatment of choice when reaction to the injury is severe. If such treatment is to be effective, early recognition of the complication is imperative.

REFERENCES

1. Butler, T. Harrison: Iridencleisis and trap-door iridectomy in the treatment of glaucoma, Brit. J. Ophth., 16:741-749 (Dec.) 1932.
2. ———: Statistics of glaucoma operations, Tr. Ophth. Soc. U. Kingdom, 38:368-375, 1918.
3. Davenport, R. C.: The after results of corneo-scleral trephining for glaucoma, Brit. J. Ophth., 10:478-484 (Sept.) 1926.
4. Dominguez, D. D.: Las causus de fracaso de las operaciones antiglaucomatosa, Arch. Soc. oftal. hispano-am., 4:575, 1944.
5. Enroth, Emil: Über Katarakt nach Elliots Operation, Acta ophth., 5:113-121, 1927.
6. Gjessing, Harald G. A.: Further investigations into results of iridencleisis operations by Holth's method, particularly as regards method combined with meridional iridotomy, Tr. Ophth. Soc. U. Kingdom, 47:358-369, 1927; Acta ophth., 5:149-159, 1927.
7. Goldberg, Herman Krieger: The results of various operative procedures in acute congestive glaucoma, Am. J. Ophth., 34: 1376-1378 (Oct.) 1951.
8. Greenwood, Allen: Comparison of operations for chronic glaucoma, Arch. Ophth. 10:472-482 (Oct.) 1933.
9. Hagen, Sigurd: Holths tangentiale Sklerektomie und Iridencleisis anti-glaucomatosa, vergleichende Statistik, Acta ophth., 10:88-90, 1932.
10. Haik, George M., Waugh, Richey L., Jr., and Lyda, Wood: Sympathetic ophthalmia, similarity to bilateral endophthalmitis phacoanaphylactica, new therapeutic methods, Arch. Ophth., 47:437-453 (April) 1952.
11. Hepburn, Malcolm: Experience gained from one hundred and forty trephine operations for glaucoma, Brit. J. Ophth., 6:97-103 (March) 1922.
12. Holst, J. C.: Ergebnisse der Sclerectomia tangentialia (Holth) und der Iridencleisis subconjunctivalis (Holth) bei Glaucoma simplex im Jahrzehnt 1920-1929, Klin. Monatsbl. f. Augenh., 87:602-617, 1931.
13. Irvine, S. Rodman, and Irvine, Alexander Ray, Jr.: Lens-induced uveitis and glaucoma, part I, Endophthalmitis phacoanaphylactica, Am. J. Ophth., 35:177-186 (Feb.); part II, The "phacotoxic" reaction, 370-371 (March); part III, "Phacogenetic glaucoma": lens-induced glaucoma; mature or hypermature cataract; open iridocorneal angle, 489-499 (April) 1952.
14. Knapp, Arnold: Operative treatment of chronic glaucoma; report of two hundred successive operations, Arch. Ophth., 10: 298-301 (Sept.) 1933.
15. Lemoine, Albert N., Jr.: Glaucoma. A statistical review of 816 patients with 1,112 glaucomatous eyes, Am. J. Ophth., 33:1353-1373 (Sept.) 1950.
16. Lemoine, Albert N., and Macdonald, Alexander E.: Observation of phacoanaphylactic endophthalmitis, Arch. Ophth., o.s.53:101-118, 1924.
17. Löfgren, Signe: Die Resultate der Glaukomoperationen, Acta ophth., 10:77-87, 1932.
18. Louhela, Terttu, and Teräskeli, Hilja: On the results of different glaucoma operations, Acta ophth., 24:27-41, 1946.
19. Meek, Raymond Emory: The Elliot trephining operation for glaucoma, a procedure to make this operation less difficult and more effective, Am. J. Ophth., 31: 1232-1240 (Oct.) 1948.
20. Mügge, Felix: Die Treparationszyklodialyse als primäre Glaukomoperation, Klin. Monatsbl. f. Augenh., 112:104-113, 1947.
21. Ploman, K. G., and Granström, K. O.: An der Augenabteilung des Seraphimerlazaretts in den Jahren 1909-1927 bei Glaukom primarium ausgeführte Sklerektomien (nach Holth und Elliot) und

Iridektomien unter besonderer Berücksichtigung der postoperativen Funktion, Acta ophth, 10:54-76, 1932.

22. Quackenboss, Alex.: Sclerocorneal trephining for glaucoma. Complications and failures in one hundred cases, Tr. Am. Ophth. Soc., 13:632-639, 1914.
23. Salus, Robert: Die Zyklodialyse nebst Bemarkung über den Rückgang der glaukomatösen Exkavation, Klin. Monatsbl. f. Augenh., 64:433-509, 1920.
24. Sugar, H. S.: Cyclodialysis, a follow-up study, Am. J. Ophth., 30:843, 1947.
25. Verhoeff, F. H.: Discussion of paper by Byers, W. Gordon M.: Histologic findings in eyeballs lost through complications of sclerocorneal trephining, Tr. Am. Ophth. Soc., 16:57-58, 1918.
26. Wilmer, W. H.: Discussion on the results of the operative treatment of glaucoma, Tr. Ophth. Soc. U. Kingdom, 47:230-294, 1927.

DISCUSSION

EDMUND B. SPAETH, M.D., Philadelphia, Pa.: I am pleased that I have been given the opportunity to open the discussion on this very fine presentation of a rather important subject; that is, lens capsule injury following decompression operations for glaucoma.

This discussion will attempt to emphasize some of the essayists' conclusions through the presentation of further clinical evidence and their final conclusions as to treatment will be especially considered. In addition, one or two of their observations will be interpreted somewhat differently.

From a practical standpoint, lens capsule injury, either by direct mechanical trauma, too commonly the fault of the surgeon, or because of other factors already mentioned, has a variable incidence depending on the type of surgery done. The La Grange type of iridosclerectomy seems to present this complication most commonly. The iris inclusion operation has it least commonly. The trephining operation, unfortunately, is the one most commonly followed by a specific type of mechanical trauma due to damage to the lens by instrumentation during the surgery. Cyclodialysis should have no complication.

The essayists' conclusion, based upon the literature, that perhaps 20 to 30 per cent of intraocular filtering procedures are followed by lens opacities within a few years, should be discussed in somewhat more detail. The additional conclusion that the majority of these opacities can be attributed to the surgical procedure is also subject to controversy. It is to be expected that eyes which needed enucleation because of unsuccessful trephining surgery should include a very large number of eyes in which the reason for failure was lens damage. One must not forget, however, the enormous number of these operations which do not terminate with the eye on the table of the pathologist, but result instead in conservation of vision. Too frequently the complication is a condemnation of the surgeon and not of the type of operation done.

An astonishingly large number of cases of simple noninflammatory glaucoma, operated in the fifth and sixth decades of life, show lenticular opacities before the filtering operation is ever done. Glaucoma and lenticular opacities are conditions not necessarily related but both are common at the same period or decade of life.

When considering our basic subject, the iris inclusion operation is probably the least insulting to the globe. Because of that it has a great advantage. The trephining operation, however, has sound indications for certain situations, and one should not dismiss the operation and depend wholly upon an iris inclusion technic. An iridosclerectomy operation probably results in the best filtering cicatrix, but it can be the most damaging when complications result. That was demonstrated in a paper over twenty years ago reporting the response to a questionnaire sent out to many operating ophthalmologists. It is quite remarkable how free from late lens opacification an operated case of simple inflammatory glaucoma can be when tension was stabilized by a properly performed basal iridectomy. This simple operation, however, can be the cause of as much damage to the lens, direct or indirect, as any other glaucoma operation.

The essayists spoke of the inexorable uveitis which is an accompaniment of the inflammation secondary to lens capsule damage. There is another relationship with uveitis which must be mentioned here. It is possible that glaucoma is too frequently considered primary. Further and more careful studies might reveal the fact that a case under investigation is actually secondary and late postuveitic. There is little doubt as to the correctness of a statement made by the Hofrat Professor Fuchs that "the careful investigation of glaucoma cases uncovers a remarkably large number of cases of very likely secondary etiology." Any type of surgery in these instances may reactivate a hitherto quiet uveitis and be responsible

for the development of a complicating type of cataract. As you saw, that condition applied to some of the eyes which Dr. Christensen showed us here with his gonioscopic drawings. It is true, as the essayists said, that these opacities can be attributed to the surgical procedure, but the essential etiology for the development of the cataract is more basic than the surgical manipulation itself. Any type of trauma could do the same thing. One more statement mentions the significance of hypotony. The discusser agrees wholly with the essayists when they state that the effect of hypotony has been very much overemphasized. One must not forget that hypotony may be an accompaniment of a certain type of cyclitic inflammation and that the hypotony is symptomatic and not causative.

There is no doubt that certain anatomic changes which are not caused by mechanical damage can occur following filtration operations. Causes for these are most important for consideration. They are such factors as abrupt opening of the anterior chamber and changes which may result from this too rapid decompression. One can actually see damage at times to the zonule at the equator of the lens in the coloboma of an iridosclerectomy from this effect. Delayed formation of the anterior chamber predisposes to lens damage. Hydrops choroidae, so-called choroidal detachment, is related to a probably significant uveitis. The development of postoperative iris and lens capsule synechia is certainly a factor in causing subsequent cataract. Inadequate postoperative observation and medication—perhaps not inadequate but better described as ineffectual—after the surgery is a factor. These and others, as mentioned, should not condemn the operation so much as to show the necessity for better control of the therapy.

Now for the second part of this discussion, that of handling situations suspiciously significant, i.e., indicating trouble which might develop or is already established. The essayists are wholly correct in their desire to use early gonioscopy as an aid to early diagnosis. It is remarkable how early one may use a gonioscopic lens with impunity in the postoperative stage of any given case. Equally important is the necessity for slit lamp microscopy at that same time or even earlier. As just said, it is not at all uncommon to have a perfect operation ruined by inept postoperative care. Perhaps somewhat less common, the reverse is also seen; that is, a poorly performed operation can have a successful final result by intelligent postoperative attention. These statements are made to emphasize the role of the gonioscope and the slit lamp. The longer one delays in correcting certain factors the more serious are the end results because of the uveitis which is developing.

Last is the treatment of those cases with frank instrument damage—perhaps any type of damage—to the lens epithelium. The essayists' personal studies of the histologic specimens which they studied permitted them to give us a rather convincing conclusion. Despite the well-established surgical principle to avoid lens extraction in the presence of an active uveitis, there is no doubt that the most advisable procedure is an early extracapsular extraction of such damaged lenses. This is not the consideration of a cataract which may mature in six months, a year, or longer after former apparently successful surgical decompression. Surgery in early lens damage cases is difficult because one must operate adequately but at the same time eschew unnecessary trauma. It seems that a most important step in the operation is the attempt to remove the capsule with capsule forceps after the lens substance has been expressed. This applies to practically all instances of extracapsular extraction, but it seems to be especially important in these cases. It almost seems that an additional toxic factor is present. Lindner once made the statement, which is wholly concurred in by the discusser, that in such situations the loss of a bead of vitreous is of much less importance than the retention of a piece of the capsule.

In conclusion, lens damage is the most serious complication connected with fistulizing operations for glaucoma, and as such is to be prevented if at all possible. If it occurs it should be diagnosed early and, of greatest importance, it should be treated promptly and adequately.

JOSEPH A. C. WADSWORTH, M.D., New York, N. Y.: Dr. Christensen has presented a subject well known to ophthalmologists, but emphasis of this important work has not been so clearly demonstrated or discussed. I can add very little to Dr. Spaeth's discussion of this fine paper, but there are several small points that I should like to add as confirmation of the work of Dr. Christensen and Dr. McLean.

The so-called quiet iritis is a definite problem after intraocular operations, and such a condition can easily be attributed to small injuries to the lens at the time of the operation.

Magin (Bull. ocul. 29:685-690, 1950) studied sections of the iris removed at the time of antiglaucoma operations and found small bits of lens capsule and lens epithelium adherent to the posterior surface of the iris. He attributed the formation of cataract to these small injuries to the lens. These small breaks in the capsule of the lens can also be responsible for the release of lens substance and thereby cause a low grade iritis. If these small dehiscences in the capsule remain open, the continual contact of lens substance to the aqueous will cause the iritis to persist, but if the small openings become sealed over, as they usually do, the iritis will subside.

In addition to the inflammation as discussed above, cataract changes may develop. Along with these lens changes, there is a tendency for the lens to absorb fluids, which encourages swelling of the lens. This swelling might contribute toward the development of a postoperative myopia which is frequently seen following glaucoma operations. This myopia may vary from a half diopter to as much as 4 diopters.

Many of the complications discussed by Dr. Christensen have often been placed in the category of unavoidable sequelae of antiglaucoma operations, but with his evidence on the importance and frequency of injury to the lens, our attention can be directed to this problem in order that we might attack the situation before irreparable damage has been done.

It was a privilege to hear this paper and I consider that Dr. Christensen and his coworker have contributed a great deal toward crystallizing the facts regarding this subject that most ophthalmologists have avoided.

DR. CHRISTENSEN, Portland, Ore.: First I should like to thank Dr. Spaeth and Dr. Wadsworth for supplementing and adding to the value of the paper.

There are one or two points of contradiction. Based upon a study of the literature, we have stated that 20 to 30 per cent postoperative filtering procedures develop lens opacity within a few years. Furthermore, we went on to state that this could be attributed to the surgery. However, we did not mean to imply that all opacities were attributable to mechanical injury to the lens. There are such factors as alterations in intraocular tension and alterations in the normal metabolism of the eye which are unknown to any of us, and we feel that undoubtedly it is this alteration in the metabolism and physiology of the eye that has contributed the majority of changes in transparency of the lens.

The point we do wish to emphasize is that the incidence of lens capsule injury undoubtedly is much greater than is generally appreciated. Once this injury has occurred, the damage is far more than a matter of simple opacification of the lens or blockage of a filtering bleb. There is another mechanism that is set up specifically, intraocular inflammation, followed by all the sequelae of an inflammatory process that may lead to the eventual loss of an eye.

FURTHER OBSERVATIONS ON INTRAOCULAR ACRYLIC LENSES IN CATARACT SURGERY

HAROLD RIDLEY, M.D. (Cambridge)
LONDON, ENGLAND
BY INVITATION

I AM deeply grateful for the honor you have conferred on British Ophthalmology by generously inviting me to speak today. Medicine and surgery know no frontiers and we all realize how important are exchanges of ideas and experiences and the value of both formal and informal discussion in the advancement of our specialty.

The subject on which you would wish me to speak is surely "Intraocular Acrylic Lenses," about which I can now provide more extensive and up-to-date information and statistics, though inevitably with a concise subject some ground will be covered which will be familiar to many who have read earlier papers.

From time to time the possibility of employing artificial lenticuli had occurred to many ophthalmologists, only to be put aside as an impossibility. In the fall of 1949, however, I performed an intracapsular extraction at St. Thomas's Hospital, London, which was witnessed as usual by undergraduate students, one of whom inquired at the end why I did not replace the diseased lens by a new one within the eye. To this unknown student must be given the credit of stimulating our researches.

The disadvantages inherent with an aphakic eye corrected by an unsightly and heavy spectacle lens are well known. The image is unnaturally magnified by about one third, the lens gives good acuity only through its optical center, oblique views causing distortion and apparent displacement of objects such as steps. The useful visual field is limited, and there is no accommodation. All these disadvantages except the last, which is usually normally reduced in cataract patients, are avoided by an intraocular lens. Furthermore if the other eye still retains useful sight, binocular vision can be restored, the two eyes being partners again instead of rivals. This is a very great advantage. We have all known cataract patients who persist in using a cataractous eye, seeing only 6/24 (20/80) in preference to an aphakic lens correctable to 6/6 (20/20). Contact lenses are seldom really successful in aphakia, being difficult for cataract patients to insert and tolerate; moreover, even they magnify the retinal image by about one sixth.

The problems to be solved were three: (1) to select a transparent material which would be tolerated within the eye; (2) to determine the size, shape and refractive power of the artificial lens, and (3) to devise a method of inserting it in the eye and retaining it in position. Preliminary animal experiments were considered but were rejected as likely to be of very limited value except as a test of the tolerance of materials of which we already had enough information, mainly as a result of war injuries. Animal physiology differs from human, subjective tests are impossible and the lens specification could not be determined by this means.

Ophthalmic Surgeon, St. Thomas's Hospital; Surgeon at Moorfields, Westminster and Central Eye Hospitals.

Presented at the Fifty-Seventh Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 12-17, 1952, Chicago, Ill.

The choice of material lay between glass and methacrylate, both of which are plentiful, consistent in composition and easily and accurately worked. Both are well tolerated and set up negligible tissue reaction in the body. Even in the eye these substances were known to cause no inflammation unless in contact with a sensitive and mobile portion such as the iris, and indeed there were many instances where the presence of transparent foreign bodies had been overlooked by competent ophthalmic surgeons. The overwhelming advantage of methacrylate is its light weight, which is only about half that of glass. Imperial Chemical Industries were consulted regarding suitable material and advised use of their product Transpex I. This is a fully polymerized variety of polymethyl methacrylate in which there is no free monomer which might be gradually liberated and set up a tissue reaction. Impure forms of methacrylate, especially those which give off a faint odor, are dangerous, whereas the pure compound has never been known to cause irritation. The specific gravity of Transpex I is 1.19 and its refractive index, 1.49. In passing it may be mentioned that human lens grafts were considered, for they would not appear to be completely impracticable though having few if any advantages over synthetic compounds which are not subject to degenerative changes.

The design of the artificial lenticulus was no simple problem because the natural lens is a complex structure, the composition of which varies as between capsules, cortex and nucleus. Moreover, its dimensions are variable especially when removed from the eye or when microscopic sections are made. Its refractive index is regarded as being 1.42, but its total power has been variably estimated between +16.01 (Tscherning) and +19.11 (Gullstrand). It might be thought from these widely differing figures that considerable individual variation occurred, but our researches seem to indicate the opposite and that most human lenses are similar in power. The standard artificial substitute usually renders the refraction within about 2 diopters of that of the other eye, the disparity being insufficient to produce troublesome anisometropia. The diameter selected for the acrylic lens was 8.35 mm., about 1 mm. less than the natural, for ease of insertion and to avoid pressure on the ciliary body. Since the artificial lens might not lie in precisely the normal situation, no means could be devised for determining its curvatures except by trial on human beings. Our prototypes too closely copied the natural lens curves and proved far too strong. The first two patients, who fully understood the experimental nature of the operation, deserve my thanks for their valuable assistance, for they proved that a lens could be inserted and tolerated and the effect was good enough to allow computation of the overcorrection so that a satisfactory design was found for subsequent cases. The specification of lens used for all but the first cases is as follows:

Material	Transpex I
Diameter	8.35 mm.
Thickness	2.4 mm.
Anterior curve diameter	17.8 mm.
Posterior curve diameter	10.7 mm.
Refractive power in aqueous (refractive index 1.33)	24 diopters

A peripheral groove is cut in both surfaces before final polishing to permit the lens to be grasped with forceps.

To retain the lens in position, consideration was given to the possibilities of sutures and similar devices but it was hoped that the lens, which is only about 15 per cent heavier than the aqueous fluid

it displaced, would be supported upright between the posterior lens capsule and vitreous body behind and the muscular iris with intact sphincter muscle in front. Fortunately this hope has been justified, and where a perfect extracapsular operation has been performed, no lens has dislocated, in fact it is surprising once the eye is soundly healed how little the refraction changes. A slight movement of a powerful lens would be of great refractive moment.

The operative technic normally employed is as follows:

The patient is given one grain of phenobarbitone as a sedative and the general preoperative routine, including conjunctival culture, is observed. The usual cataract instruments are prepared with the addition of a pair of lens-introducing forceps and a special iris hook. The lenses, for there should always be a spare in case of accidental slipping, are placed in a special metal rack and sterilized for one hour in cetrimide 1 per cent after which they are thoroughly washed in distilled water. Temperatures over 70° C. must be avoided, for they cause distortion of the acrylic material. The lenses are set in the rack correctly oriented so that the flatter front surface is known to the surgeon before the operation begins.

The pupil should be dilated to at least 5 mm. diameter with homatropine only. Full dilatation is by no means essential or desirable, for the curved edge of the acrylic lens will widen the iris aperture. Procaine 4 per cent is injected over the neck of the mandible to block the upper branches of the facial nerve which supply the orbicularis muscle, and a simple lid stitch is inserted to insure closure of the eye after operation. Anesthesia is provided by 4 per cent cocaine drops only, no retrobulbar injection being required.

Two half-depth corneoscleral mattress sutures are inserted and an otherwise normal cataract section is cut with a Graefe knife including where possible a small conjunctival flap (the writer does not in ordinary extractions find any sutures desirable). The anterior capsule is incised as far down as possible with a cystotome, and very careful and complete removal is effected with toothed capsule forceps. After expression of the lens nucleus all cortical remnants are removed by thorough irrigation with normal saline. It is sometimes necessary to insert smooth capsule forceps again to remove loose remnants. If it is decided that the posterior capsule has been completely cleared, the acrylic lens may be inserted forthwith; if not, it is advisable to close the eye, leaving it with an ordinary extracapsular extraction. In point of fact I have never so far had to do this, though complete removal of cortical and capsular remnants has sometimes called for considerable perseverance and prolonged irrigation. In several cases, however, I have reopened an eye in which a traumatic cataract has absorbed or on which another surgeon has performed a perfect extracapsular extraction and inserted an acrylic lens at a second stage some months after the extraction. Assuming that the posterior capsule has been cleared, the acrylic lens is grasped by its peripheral groove with the special forceps and removed from the lens holder with its anterior surface upwards. The corneoscleral section is opened by an iris repositor held in the surgeon's other hand, and the acrylic lens is guided into the eye downwards and backwards with a slight side-to-side movement so that it enters the pupil and lies partly beneath the lower portion of the iris. The forceps can then, and only then, be relaxed, and the lens is steadied by the repositor while the sides and upper portion of the iris are manipulated over it with the point of a hook or cystotome.

Final centering is effected by external pressure on the cornea and sclera by iris repositors. A small peripheral iridectomy should be made, and after a final irrigation of the anterior chamber, the corneoscleral sutures are tied. Penicillin is instilled; it is not considered generally necessary or advisable to use miotics, and both eyes are padded for 48 hours.

The patient is allowed out of bed on the third or fourth day and the sutures are removed on the eighth day. Even pressure by the muscular iris, almost comparable to gravity, keeps the light lens securely upright in the patellar fossa.

Some degree of mild plastic iritis is to be expected for a few weeks after operation. The exudate, colored by iris pigment granules, adheres to the lens surface, though not to the cornea, but eventually clears, apparently in all cases. Clearing can be appreciably hastened by local application of cortisone. In some cases weak mydriatics are used, but care must be taken not to dilate the pupil fully for fear of the lens dislocating into the anterior chamber.

It is quite essential that the cataract extraction should be perfectly performed and the pupil left central and circular if an acrylic lens is to be inserted. The patient should not be younger than 12 years of age, when the eye is practically fully grown, nor older than 75, though some good results have been obtained in those over 80. Myopes are generally unsuitable; the risk is rather greater and a cataract glass weaker than usual would normally be required.

Intracapsular extraction has been performed in some 10 cases. Usually this has been accidental, capsule forceps being employed after extraction and irrigation to remove tags of anterior capsule have carried away the posterior capsule as well. In other cases a deliberate intracapsular extraction has been planned, with considerable trepidation, because the cataract has been an immature posterior cortical or nuclear type and the patient has demanded an acrylic lens. In this operation the acrylic lens must be slid over the vitreous face. I fear this procedure, for it is clearly both difficult and dangerous, and can recommend it only to operators more proficient than myself. It is important in intracapsular cases that the pupil should not be fully dilated; if so, the lens cannot be retained beneath the lower part of the iris while the upper is manipulated for the vitreous dome tends to push it forward.

The results of the first 63 cases seem to me to be most encouraging and I cannot help wondering when our good luck will change. The first 27 cases have already been reported. The first 2 form a series of their own because the lens employed was too large and too powerful. Of the other cases reported in February 1952, in the preceding December, 2 patients saw 6/36 (20/120) letters, 1 saw 6/24 (20/80), 3 saw 6/18 (20/60), 2 saw 6/12 (20/40), 5 saw 6/9 (20/30), 5 saw 6/6 (20/20) and 2 saw 6/5 (20/15). With two exceptions these eyes nine months later see as well or better than before. Now 4 see 6/18 (20/60), 4 see 6/12 (20/40), 3 see 6/9 (20/30), 6 see 6/6 (20/20) and 3 see 6/5 (20/15). The exceptions are one unsuspected monocular myope, whose lens slipped after an intracapsular operation, and one boy, who returned to hospital with a black eye after a fight, with the acrylic lens partly in front of the iris. Both eyes are, however, quiet.

Thirty-six cases have undergone the operation at St. Thomas's Hospital and Moorfields Eye Hospital since the beginning of this year. In 2 cases both eyes contain acrylic lenses, but the last 14, since June 30, are too recent to record. Of the remaining 22, which under-

went operation in the first half of 1952, 1 sees 6/18 (20/60), 3 see 6/12 (20/40), 9 see 6/9 (20/30), 3 see 6/6 (20/20) and 2 see 6/5 (20/15) at the present time and several may be expected to improve later, as did the earlier series. It is fair to say, therefore, that at present over 50 per cent of our cases see 6/9 (20/30) or better, and more than half of these see 6/6 (20/20) or better; 2 eyes read 6/5 unaided. These visual acuities, moreover, are obtained without the unnatural magnification provided by ordinary cataract glasses. The patients have, of course, few of the disadvantages of aphakia, and their two eyes are partners providing binocular vision.

It always seems that one learns, or should learn, more from the misadventures, difficulties and failures than from cases which proceed uneventfully to a happy and successful conclusion. I propose therefore to discuss all the complications I have met in the hope that others may benefit from and avoid my mistakes.

The first 2 cases, in which the acrylic lens was too large and too powerful have already been reported and form a preliminary series of their own, and the next 2 had lenses of the correct refractive power but too great thickness. Among the extracapsular cases two lenses have subluxated, part of the prosthesis becoming anterior to the iris for the good reason that the preliminary extraction, in both instances preceding the lens insertion by several months, was imperfect. In one of these a portion of the iris root was left adherent to the Graefe section so that about one fifth of the lens circumference is visible. In the other a hole in the iris caused by an intraocular foreign body was found to be adherent to the underlying capsule when the acrylic lens was inserted. Had this been foreseen, an attempt would have been made to divide this synechia some days before. As it happened, the lens was inserted but was held up by this adhesion, making the pupil vertically oval, but the acuity was 6/9 (20/30). Six months later the patient, a boy of 12 years, was involved in a fight and returned with a black eye and a partially dislocated lens which it proved impossible to replace permanently. The eye however is fairly quiet and the acuity now 6/36 (20/100), a great improvement on a unilateral aphakic eye.

In one case, a rather feeble old man of 76 years, the section failed to heal in the normal time and gaped on removal of the corneoscleral sutures. This led to slight iris inclusion, the formation of a false filtration angle and glaucoma. One very difficult old woman aged 84 ruptured her section on the fourteenth day and the presenting lens was grasped and removed with forceps. Two cases developed a moderate rise of tension associated with a shallow anterior chamber and narrow filtration angle but both readily responded to miotics.

One early case required division of the posterior capsule from behind. One iris prolapse required repositioning.

Apart from an invariable and temporary deposition of fibrin and iris pigment on the anterior lens surface, sometimes preventing good vision for a considerable time, the extracapsular cases have given no other trouble.

Among the smaller number of intracapsular cases there have been two total dislocations of lens into the bottom of the vitreous chamber. Both these cases were found to be highly myopic eyes and as they remained quiet the patients were rather pleased with the unexpected development. In one of these cases the vitreous face may still be seen flat and unbroken behind the pupil, the other has a little vitreous in the anterior chamber.

In three other cases, two of which were traumatic cataracts, I have felt

unable to insert an acrylic lens because my extraction was imperfect. For these failures, however, the acrylic lens cannot be blamed since its insertion was not attempted. One other unfortunate case is instructive. Hypermaturity and a tough capsule caused an inadvertent but perfect intracapsular extraction. The pupil was dilated so fully that I could not retain the lens beneath the narrow iris owing to pressure from the vitreous dome. I know now that I should have waited for contraction of the pupil by miotics before inserting the lens. The patient was very disappointed at being left with a plain extraction though the eye sees 6/5 (20/15). Too large a pupil is a disadvantage.

This list of difficulties and complications may at first seem alarming but I must affirm that it is complete. Nearly 90 per cent of the cases proceeded uneventfully, a proportion which I feel is indeed fortunate in view of the novelty and difficulty of the technic. Let us now consider the brighter side.

Sympathetic ophthalmia or prolonged iridocyclitis have not been encountered.

Serious glaucoma, except in one total failure, has not occurred.

Capsulotomy has been required in only one case. The pressure of the lens stretches the extremely thin posterior capsule, a simple membrane only 4 microns thick centrally, and prevents wrinkles which are the usual cause of capsule trouble in otherwise perfect extracapsular aphakia.

Fibrin and pigment deposited on the lens initially always clears in weeks or months and absorption is accelerated by cortisone.

The refraction is usually within 2 diopters of that of the other eye, so that binocular vision is regained with perhaps the aid of a low power lens.

The patients' reactions to this operation are interesting. None have complained of any abnormal sensation indicating the presence of a foreign body in the eye. No iris pain seems to be produced and strange to relate no late iridocyclitis has occurred. Patients appreciate binocular vision from the start even when the acuity of the operated eye is somewhat dimmed by exudate on the lens. Some seem to think that the sight of the other eye has been improved. The synoptophore has been employed in a number of cases to confirm the presence of single binocular vision. The only symptom worth reporting is that several complain of seeing red in bright sunlight or when snow is on the ground probably because the artificial lens absorbs less long-wavelength light rays than the natural. A strange finding is that the depth of focus seems greater than expected though clearly no accommodation is possible. One eye read unaided 6/5 (20/15) partly and 6/5 (20/15) fully with -0.5D sphere and also Jaeger 6 at two feet.

My first paper on this subject was presented at the Oxford Ophthalmological Congress over a year ago when our first artificial lenticulus had been in the eye only one and a half years. Had the handful of my colleagues who alone knew of our researches not encouraged me I should have waited longer before publication. I said at that time that great caution was advisable and I still think caution is desirable for many years to come. Nevertheless I must admit that my confidence has become much greater in the past year. Surgical technic has improved and many excellent results have been obtained. The most encouraging feature, however, is the finding that the lens is well tolerated and that the early cases are quieter than before. In many eyes which have contained acrylic lenses for more than one year the lens surface is as clear as when it came out of the sterilizing solution, and

in fact, it is quite difficult to see it with the slit lamp.

It now seems probable that an eye containing a satisfactory acrylic lens is actually stronger and safer than an ordinary aphakic eye. The lens acts as a bulkhead rendering iris prolapse unlikely and keeping the vitreous in place, so lessening the possibility of retinal detachment and symptoms such as floating spots associated with vitreous shrinkage and detachment.

Time alone will show, but I now feel hopeful, that this operation will be widely employed to complete the cure of cataract which is only half treated by extraction. One day, no doubt, science will find means to prevent rather than cure senile lens opacities.

My thanks are due to my friend, Mr. J. Pike of Rayners for his help in designing and manufacturing the acrylic lenses and to the photographic department of St. Thomas's Hospital for the lantern slides. Colonel F.R.B. Skrimshire again determined the refraction and acuity of nearly every case. Last but not least I thank my early patients for their courage and the trust they put in me.

BIBLIOGRAPHY

1. Arruga, A.: Problemas immediatos de la inclusion de la lente de Ridley, Arch. Soc. oftal. hispano-am., 11:1490-1507 (Dec.) 1951.
2. Ridley, H.: Intra-ocular acrylic lenses, Tr. Ophth. Soc. U. Kingdom, 71:617-622, 1951.
3. ———: Artificial intra-ocular lenses after cataract extraction, St. Thomas's Hosp. Rep., 7:12-14, 1951.
4. ———: Intra-ocular acrylic lenses after cataract extraction, Lancet, 1:118-121 (Jan. 19) 1952.
5. ———: Intra-ocular acrylic lenses; a recent development in the surgery of cataract, Brit. J. Ophth., 36:113-122 (March) 1952.
6. ———: Further observations on intra-ocular acrylic lenses, Tr. Ophth. Soc. U. Kingdom, (In press.)

DISCUSSION

DERRICK VAIL, M.D., Chicago, Ill.: We are greatly obliged to Mr. Ridley for giving us at first hand the latest information on his extraordinary method of replacing a cataractous lens by an acrylic one. Most of us in this country first heard of it through an article in Time and through press releases carried by nearly every paper, I suppose, in this country. The medical articles in our lay press are not infrequently open to suspicion and skepticism.

The passage of time, alas, has only too frequently justified our suspicions. Reputable ophthalmologists are harrassed and bombarded by questions from their patients and often find it most difficult to give satisfactory and fair answers to these questions, which after a further passage of time no longer need to be answered, one way or the other.

In this case, however, those of us who were interested were soon able to read the original article published in The Lancet on January 19, 1952, and could not help but be influenced by the sterling reputation of the author and the scientific integrity of The Lancet. Many of us were deeply concerned then and a lot of us still are concerned. Our first reaction was one of amazement at the ingenuity of the idea and the courage and skill of its originator. There are many of us here who still believe, I am sure, in spite of Mr. Ridley's remarkably successful run of cases here reported, that the operation is one of considerable recklessness and that its hazards far exceed the little that is gained in the way of ocular comfort to the patient and the questionable advantage of binocular vision obtained at such an obvious risk.

Until further work is done and more time has elapsed and more of the risks involved I do not want such an operation performed on myself nor can I advise it for my patients, willing as they might be to undergo the additional hazards of which they can have no true conception. I believe that this is a question that must be honestly and courageously faced by each ophthalmic surgeon.

It is, of course, entirely possible that time will show that Mr. Ridley's operation does not add any new or more serious complications directly induced by the insertion of a foreign body into the eye to the already long list, too familiar to us all, of, shall I say, the old fashioned cataract operation. These complications give us enough concern as it is, and sometimes keep us awake at night.

I am fully aware that by these remarks I expose myself to the charge of being opposed to progress. Those who opposed the use of sutures in cataract surgery and those who opposed the intracapsular extraction of the lens are now almost forgotten and certainly are few in number at the present time. But their opposition, I think benefited the cause of sutures and intracapsular cataract extraction by interposing much needed caution and conservatism. I am agreeable to be called a reactionary in this instance today and hope that my efforts here will prevent, or at least retard, the recklessness of inexperience and the self-advertising viciousness of expediency. At the same time these are most exciting experiments performed on willing human subjects and I shall calmly await the reports of further work in this extraordinary field by Mr. Ridley and other ophthalmic surgeons of similar integrity and skill, and the inevitable crucible of time.

T. N. Hamdi, M.D., Philadelphia, Pa.: Last spring Dr. Warren Reese and I became aware of the existence of the Ridley implant, and our impression was very much the same as that of most of our colleagues—that it was a very bold thing to do. However, we got in touch with Mr. Ridley by mail and we found that his good results apparently continued even after he had written his article. We got the necessary extra instruments and last March at Wills Eye Hospital we did the first two operations. We have done a number since then. We have attempted the operation in 11 cases. In one of them we found the pupil was so large as a result of the inadvertent use of atropine that we did not place the lens in position because we knew it would not stay. Another case was not done because we found there was a great deal of residual cortex remaining. We felt that the presence of the lens would interfere with its absorption. The other 9 cases received the implant.

With the first two operations we had much trial and tribulation mentally because we realized that there was a great deal at stake. We found that about the fourth day postoperatively there was what appeared to be in the slit lamp a tremendous anterior uveitis. There did not appear to be any keratic precipitates, but there was a dense flare and a lot of plastic exudate which formed what appeared to be a complete synechia of the iris. We were very thankful at that point that there was a good, adequate peripheral iridectomy, which, I am sure, prevented a very severe secondary glaucoma.

After about ten days, with the use of cortisone and with very careful use of homatropine—we were still very much afraid the lens might pop out—the uveitis had subsided. We found that the pupil was just about covered by dark brown pigment deposition and exudate. This depressed us because we felt that the patient would not be able to see through that. But as several weeks went by, we found that this pigment became smaller. It seemed to shrink and part of it I am sure must have dropped off, leaving a very adequate area of clear lens exposed for vision.

Our first two patients when last examined some weeks ago were able to see 6/12 without any aid. However, it was not possible to improve their vision better than 6/9.

The operations which we have done subsequently we have done with more facility because we have had more experience. We find that it is possible to do this operation rather easily. The original fumbling that we noticed in putting in the lens has been avoided. Of course, there is a certain amount of luck, and when you have done only 9 operations you cannot say that you are developing any skill. It may be that we just had a few lucky breaks, but I do feel that in the last couple of cases the lens seemed more or less to "find" its position in the patellar fossa. We have had less postoperative reaction, probably because we now use atropine and cortisone routinely.

The first two cases had 6/12 vision; the third case had 6/6, with proper correction and without any glass at all, read Jaeger 1. He drives his car and has excellent binocular vision with his fellow eye, which is 6/6. Of the other cases, there was one 6/6 and the others were either 6/9 or 6/12. I don't remember them exactly. Lest you feel that they were all uniformly good, there was one patient who developed a very severe uveitis when she was not immediately available to us for treatment. She took a trip to an adjoining city in an automobile and was exposed to a draft. At least, she felt that was what did it. We know that she was in good shape when she left our office several days previously. But there was a severe uveitis, and the lens apparently was not exactly in its proper position for some reason. On later slit lamp examination we found that the iridectomy margins had become plastered onto the lens.

The eye is now quiet and the tension is normal, but there is still so much exudate on the lens that we cannot tell what the patient will see. We are waiting, hopeful that the same thing will happen here that happened with the other cases, namely, that the exudate will shrink and perhaps drop off.

There are certain features that Mr. Ridley did not mention that perhaps would be important to mention now. One is that we have purposely chosen our cases with extreme care. We have not followed this procedure on anyone who had a history of glaucoma in the family. We have not done it, needless to say, where there was a personal history of glaucoma. We have examined them very carefully both in their history and with the slit lamp, for any signs of tendency to uveitis. And also we have tended to do this operation on those patients who would be benefited by the binocular vision—in other words, those who had a good fellow eye. We have done it on several patients who had a bad fellow eye because we felt they would benefit by the other features touched upon by Mr. Ridley.

Mr. Harold Ridley in reply thanked those who had discussed his paper. He appreciated Dr. Derrick Vail's note of caution. Though many excellent results had been obtained, the speaker still selected his cases carefully. He had performed only 70 acrylic lens operations in three years though some 600 cataracts had been under his care in that period. Any complicated cataract or gross constitutional disease, such as diabetes, was a contraindication, and the most suitable cases are those in which one eye contains a mature cataract and the other a clear lens. An ophthalmic surgeon with a unilateral cataract had asked to have an acrylic lens inserted in his own eye when the speaker returned to London.

Postoperative iritis was the only frequent complication and there was a suggestion that this might be due to traces of the sterilizing fluid remaining on the lens rather than to the material itself, for it was always temporary and did not recur. So long as precision surgery had been performed and the lens was in place behind a central and circular iris he felt confident that the final result should be satisfactory. He was now dissatisfied if the acuity could not be improved to at least 20/30. Sometimes the patient can see faces clearly only one day after the cataract operation.

The acrylic lens operation must be performed with close attention to detail. It is not an operation for every case and perhaps not for every surgeon. The speaker found great satisfaction in hearing colleagues who had never performed or seen an acrylic lens operation say it was impracticable, later become interested and perform one and finally became quite enthusiastic. He wished the assembly good luck with any cases they undertook.

A CASE OF MULTIPLE MALIGNANT MELANOMAS IN ONE EYE

DAVID A. ROSEN, M.D.
and
GARDNER N. MOULTON, M.D.
BALTIMORE, MD.
BY INVITATION

A CURSORY review of the ophthalmic literature reveals that there have been exceedingly few reported cases of multiple primary malignant melanomas of the choroid.

Fuchs,[1] in a monograph on tumors of the eye written in 1882, mentioned nine cases in which more than one tumor mass was situated in the choroid. A review of these cases demonstrates that only one of the instances fulfills acceptable criteria for multiple primary tumors, although the data submitted is admittedly brief and incomplete. Three of his cases occurred in young children. One case occurred in an eye harboring a wooden foreign body.

Velhagen,[2] in 1920, reported on two juxtaposed nodular malignant melanomas in a single eye. Although the intervening choroid was felt to be free of tumor, the sclera between the two lesions was found to be infiltrated with tumor cells. The evidence would thus appear to be against this case being one of multiple primary melanomas of the choroid.

Rochat,[3] in 1926, reported on an instance of two malignant tumors of the choroid of equal size occurring in one eye in a forty-year-old man. In this case, the second nodule was noted one year after the first observation of a fundus mass. After enucleation, the two lesions were found to be separated by a distance of 11 mm., and the intervening sclera and choroid were found to be normal. Serial sections, however, were not made. The patient was alive and well four years after the first tumor was discovered.

In his recent volume on tumors of the eye, Reese[4] states that he knows of no authenticated cases on record of multiple primary malignant neoplasms of the choroid in a single eye.

We have recently had the opportunity to study an eye after an enucleation containing multiple separate malignant melanomas of the uveal tract. At least two of the lesions are completely independent, as proved by serial sections. It was, hence, felt worth while to report this case. We report it through the courtesy of Dr. William C. Owens.

CASE HISTORY

Mr. J.R., a 52-year-old white male, first came to the attention of an ophthalmologist in 1947. He was found at that time to have a lesion in the left eye which was considered by the examining physician to warrant admission to the hospital. This advice was not heeded. During the ensuing eighteen months the patient gradually lost the central vision in the left eye. Four years later, in September 1951, the patient first presented himself to Dr. Owens, complaining of difficulty in focusing with his right eye. There had been no pain or redness of the left eye.

Examination of the left eye revealed a large mass extending out from the disc to the far temporal periphery. The mass was found to be elevated 6 diopters, and to have steep margins. A few striae were recognized in the retina below. In the extreme temporal periphery a bullous detachment was noted. The mass barred transillumination. The intraocular tension was normal.

From the Wilmer Ophthalmological Institute of the Johns Hopkins Hospital and University.
Presented as a *Clinicopathologic Case Report* at the Fifty-Seventh Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 12-17, 1952, Chicago, Ill.

The patient was admitted to the Wilmer Ophthalmological Institute of the Johns Hopkins Hospital. General physical examination, except for the left eye, was completely normal. No evidence was found of other primary tumor, or of metastatic tumor. On September 16, 1951, the left eye was enucleated. At the time of enucleation no extrabulbar extension was found.

At the time of this writing the patient is still in excellent health.

Pathology

Gross Examination. The eye is normal in size and shape. There is spicule-like pigmentation of the sclera temporal to the optic nerve. There is a large tumor mass, 15 mm. x 17 mm. in size, extending from the region of the macula to the ora serrata. This mass is relatively flat and is grayish in color. Four smaller flat discrete tumor nodules, varying from 2 to 8 mm. in diameter are seen about the optic nerve head. There is a small simple detachment of the retina about the largest tumor.

Microscopic Examination. Anterior segment: The cornea and iris are normal. The anterior chamber is shallow. One chamber angle is filled with spindle-shaped tumor cells which are continuous with a flat mass involving all portions of the ciliary body on one side. This mass is composed of spindle cells of Callender type A which are largely arranged in parallel bundles. The portion of the tumor in the pars plana is more heavily pigmented than that in the corona ciliaris. There is invasion of the overlying sclera. Beyond the ciliary body tumor there is a short span of normal choroid and sclera, after which a large choroidal mass is encountered. This mass is composed entirely of spindle cells of Callender type B, and is only slightly pigmented. There is moderate vascularization of the tumor, and there is no necrosis. There is degeneration of the overlying retina, and in one place the retina is penetrated by the tumor.

Posterior segment: Sections made through various portions of the posterior segment of the globe reveal a number of discrete malignant melanomas of the choroid, varying in histologic type. Spindle cell type B is predominant; however, epithelioid and mixed cell types are present. The actual number of lesions cannot be stated inasmuch as serial sections were not made through the whole eye. However, serial sections carried through a portion of the posterior segment which appeared grossly to contain a single moderate sized tumor of the choroid, reveal two separate and distinct malignant melanomas of the choroid which are in no way connected with any other lesion. Furthermore, a single benign melanoma of the choroid is encountered in one of the sections. There is invasion of the sclera over the individual masses, but no penetration is present. In many places the overlying retina is invaded by tumor. In no instance is intravascular extension of the tumor encountered.

The remaining pathology in the eye consists of optic atrophy, papilledema, and a hyaline body in the optic nerve anterior to the lamina cribrosa. There is cystic degeneration of the retina, and a simple detachment of the retina in relation to the individual tumors. Numerous drusen of Bruch's membrane are noted.

Pathologic diagnosis: Multiple malignant melanomas of the choroid; malignant melanoma of the ciliary body; benign melanoma of the choroid; simple detachment of the retina; hyaline body in the optic nerve head; papilledema, and optic atrophy.

COMMENT

The association between malignant melanomas of the uveal tract and benign melanomas of that tissue is well known. The coexistence in one eye of more than one malignant melanoma of the uvea, each of which arises as a primary growth, however, appears to be exceedingly rare, as judged by the paucity of reported cases. Multiple malignant lesions are well known in the iris, but here a seeding out of tumor cells onto the iris surface is the postulated mechanism.

The case reported above would appear to be unique. The eye examined has several malignant melanomas of the choroid, at least two of which are proved by serial sections to be completely independent. The ciliary body melanoma also appears to be separate from any choroidal lesion. The exact number of lesions cannot be stated in the absence of serial sections through the whole eye. However, the gross observation of five separate tumors in the choroid added to the ciliary body tumor, which was not observed grossly,

brings the number of lesions to at least six. Since serial sections through what appeared grossly to be a single lesion revealed two independent lesions, one might surmise that even more separate tumors are present in this eye.

Against the possibility of this case being one of multiple metastases to the uvea is the fact that none of the lesions are in relationship with, or appear to arise from, blood vessels. Moreover, the pathologic condition noted is identical to that of ordinary malignant melanomas of the uveal tract. Finally, there is no evidence of a primary tumor or of metastatic tumors elsewhere in the patient.

REFERENCES

1. Fuchs, E.: Das Sarcom der Uvealtractus, Wien, 1882.
2. Reese, Algernon B.: Tumors of the Eye, New York, Paul B. Hoeber, Inc., 1951, p. 241.
3. Rochat, G. F.: Zwei gleichgrosse Sarkome in einem Auge, Klin. Monatsbl. f. Augenh., 76:651-655, 1926.
4. Vellhagen, C.: Ueber den Befund von zwei Choroidalsarkomen in einem Augapfel, Klin. Monatsbl. f. Augenh., 64:252-255, 1920.

NEW SCISSORS FOR ENLARGEMENT OF CATARACT INCISION

RAMÓN CASTROVIEJO, M.D.
NEW YORK, N. Y.

IN the September 1952 issue of the American Journal of Ophthalmology, Herbert M. Katzin describes a new cataract scissors. In a footnote credit is given to Harvey Thorpe and Ramón Castroviejo for similar or identical scissors. These scissors of Thorpe and Castroviejo mentioned by Katzin were designed by me and made by Storz Instrument Company* of St. Louis, Mo. over two years ago. Katzin's scissors although resembling mine are neither similar nor identical. My scissors are a modification of the scissors used for over twenty years in square corneal transplantation and have the following characteristics, as compared with the scissors of Katzin.

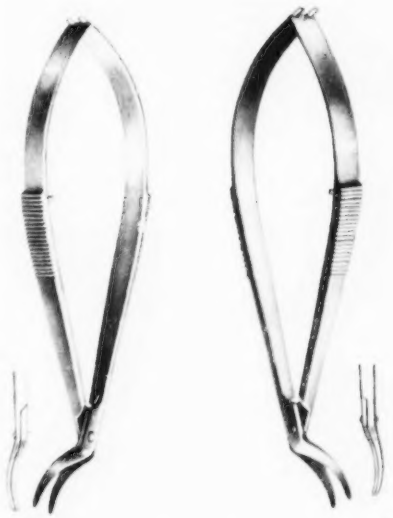


FIGURE 1.

They are used as a pair, right-hand and left-hand curves; spring handle action; the blades are angulated in relation

From the Ophthalmological Department of St. Vincent's Hospital and the New York University School of Medicine.

Presented as a *New Instrument* at the Fifty-Seventh Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 12-17, 1952, Chicago, Ill.

* Scissors are manufactured by the Storz Instrument Company, 4570 Audubon Avenue, St. Louis, Missouri.

to the spring handle to facilitate easy manipulation. The curvature of the blade conforms to the curve of the limbus, thus permitting the accurate enlargement of the incision after the anterior chamber has been entered by either keratome or scissors. The blades are one-third shorter than those of Katzin, making it easier to cut more accurately with them. The points in mine are blunt to prevent injury to intraocular structures. Those of Katzin are sharp pointed. In mine the blade which is introduced into the anterior chamber on both right-sided and left-sided scissors is located posteriorly, permitting cutting the section accurately as may be desired, either with a bevel or at right angles. In the only Katzin model which I was able to observe, the left-sided scissors has the blade which enters into the anterior chamber located posteriorly, but the right-sided scissors has this blade located anteriorly, which is an undesirable position for the blade in order to cut accurately.

The new scissors (fig. 1) that I am now presenting have been tested by me and other ophthalmologists for the past two years and have been found to be entirely satisfactory for enlarging the cataract incision.

I would like to express my appreciation to Dr. Katzin for his kindness in mentioning my scissors in his article.

IMPROVED MOTORIZED INSTRUMENT FOR DACRYOCYSTORHINOSTOMY AND CATARACT EXTRACTION BY SUCTION

RAMÓN CASTROVIEJO, M.D.
NEW YORK, N. Y.

AT THE 1951 annual meeting of the American Academy of Ophthalmology and Otolaryngology I presented a motorized instrument of small size and moderate cost that could be used for dacryocystorhinostomy and cataract extraction by suction. This type of motorized unit has been further improved both in appearance and performance (fig. 1, A and B). The improved instrument consists of a motor with 0.1 H.P., AC-DC current, 110 volts and 60 cycles. The flexible shaft which was used on the original model and found to be impractical because of excessive vibration has been replaced with an all cord driven dental arm (a), which gives considerably more range of movement, and vibration is reduced to a minimum. This arm is fitted to a Chayes (b) stainless steel hand piece which permits easy insertion of the burr used to perforate the bone. The arm and hand piece can be easily removed from the model for sterilization. On the opposite end of the motor to which the dental arm is attached is the suction mechanism which will furnish 65 cm. of vacuum at sea level. The amount of vacuum shown on a gauge (c) can be easily regulated by a thumb screw vaive (d). The rubber tubing provided with the suction cup or eresiphake is easily connected to the suction valve control (e). Although the unit is supplied with a simple handle with suction cup or eresiphake (f), any other conventional type of eresiphake may be used. The unit is supplied with a foot control rheostat (B, a). When the instrument is used for dacryocystorhinostomy, the suction mechanism may be

The instrument is manufactured by the Storz Instrument Company, St. Louis, Mo.

From the Ophthalmological Department of St. Vincent's Hospital and New York University School of Medicine.

Presented as a *New Instrument* at the Fifty-Seventh Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 12-17, 1952, Chicago, Ill.

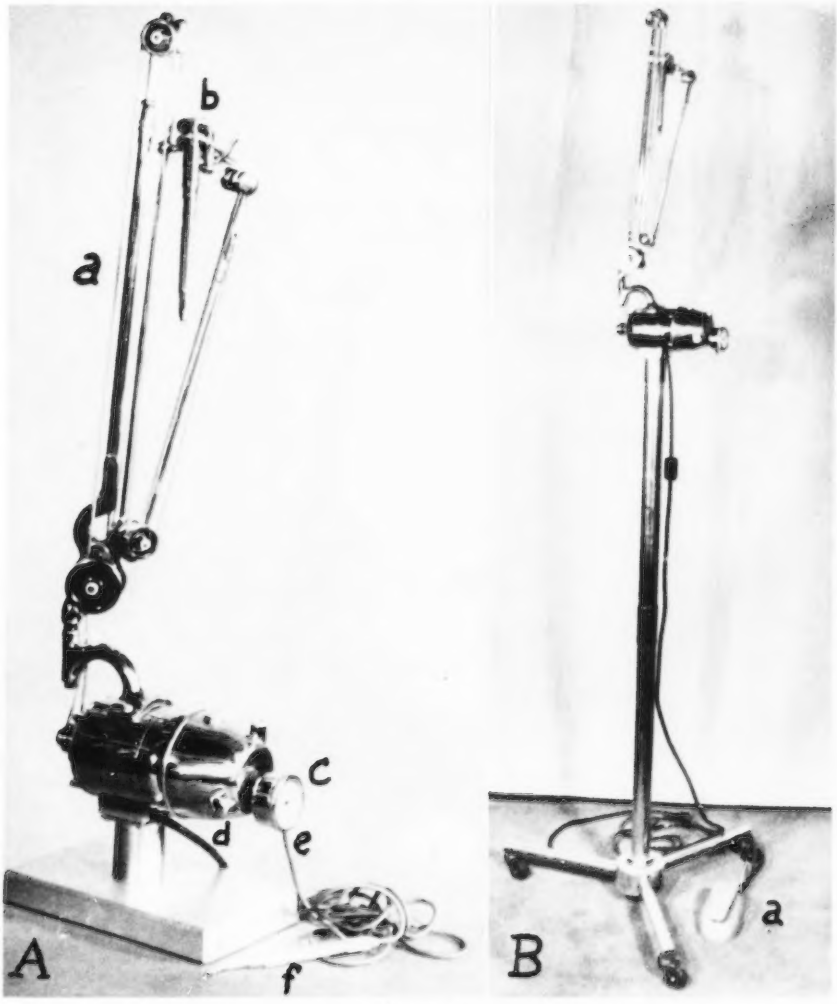


FIGURE 1.

used through an intermediate bottle to keep the operating field free from obstructing hemorrhages. When the unit is used only for cataract extraction, the dental arm may be entirely removed. The motor is supplied with two mountings, a compact rectangular base for table use (A) and a floor stand mounted on three legs with casters (B) all chrome plated. The entire unit can be disassembled to be packed compactly in a carrying case.

CHART FOR TESTING NEAR VISION

WALTER H. FINK, M.D.
MINNEAPOLIS, MINN.

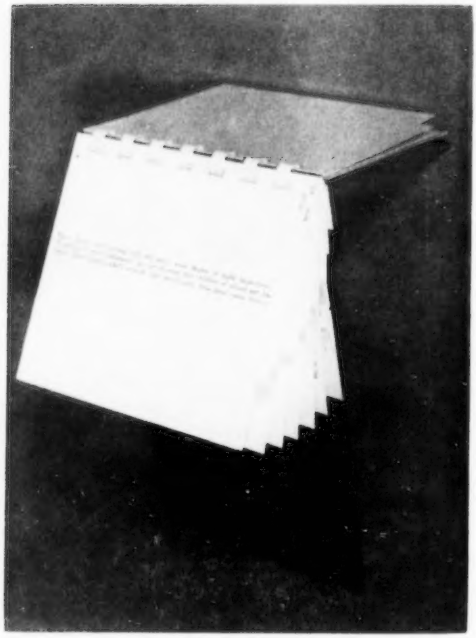


FIGURE 1.

THE READING chart consists of a series of typed paragraphs, each of which differ in size. One paragraph is exposed at a time, thus avoiding the confusion which results when a chart has several paragraphs.

The visual acuity and percentage of vision are indicated in one corner of the chart (fig. 1). These are placed in reverse so that the patient cannot read them. The various charts have the standard gradation in size of the print.

Also included is a chart for children, portion of a telephone directory, and music score.

Presented as a *New Instrument* at the Fifty-Seventh Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 12-17, 1952, Chicago, Ill.

SUPERIOR OBLIQUE TUCKER

WALTER H. FINK, M.D.
MINNEAPOLIS, MINN.

THE INSTRUMENT is designed especially for tucking the superior oblique (figs. 1, 2 and 3). As it is a reverse action instrument, the instrument handle is compressed in applying it to the oblique tendon. When the tendon is in place on the arm of the instrument, the pressure on the instrument is released and the tendon is drawn through the opening of the tucker. The amount of tuck can be regulated by a setscrew.

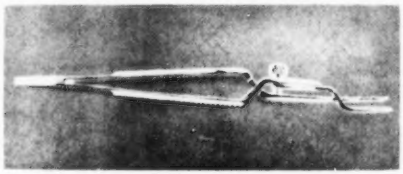


FIGURE 1

The instrument has the advantage of being smaller than the regulation tucker and of such design that this technically difficult step is made more simple than when the regulation tucker (which is designed for the horizontal muscles) is used.

Another important advantage of the instrument is that because of its design, the tuck can be placed closer to the oblique insertion.

Presented as a *New Instrument* at the Fifty-Seventh Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 12-17, 1952, Chicago, Ill.

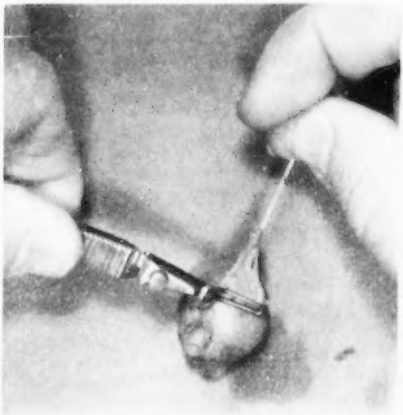


FIGURE 2

FIGURE 3

OPERATING LIGHT
(Modified Ortho-Lite)

WALTER S. ATKINSON, M.D.
WATERTOWN, N. Y.

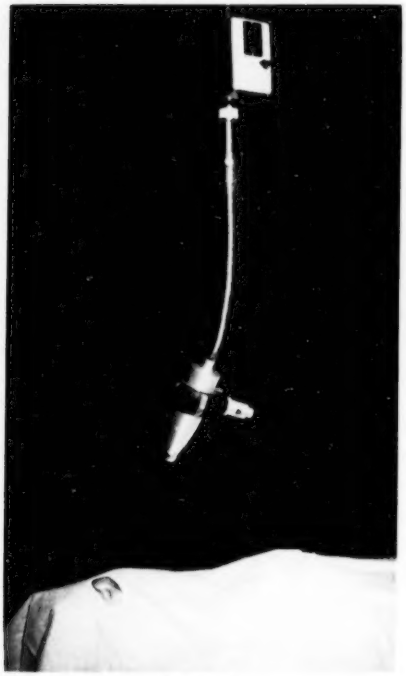


FIGURE 1.

FIGURE 2.

AN Ortho-Lite which is attached to the ceiling or, better still, from the track of a general operating room light is out of the way (figs. 1 and 2). When attached to the track of the operating light it can be moved laterally over a large arc, which is desirable.

It differs from the Ortho-Lite floor model by having the cable enter the end rather than the side of the light.

When covered with a sterile towel it can be adjusted by the surgeon. Several years of use have proved it to be satisfactory.

Ortho-Lite—Made by Bausch & Lomb Optical Co. Presented as a *New Instrument* at the Fifty-Seventh Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 12-17, 1952, Chicago, Ill.

A MODIFIED CYSTOTOME TO MAKE THE INCISION FOR PREPLACED SUTURES

WALTER S. ATKINSON, M.D.
WATERTOWN, N. Y.

FIGURE 1.

IT IS OFTEN difficult to make a clean incision of the proper length and depth for preplaced sutures with a keratome, Lundsgaard, or Bard Parker knife. This is particularly true if the eye is soft. A modified cystotome in which the blade is 0.75 mm. to 1.0 mm. in depth and the shaft is bent at 45 degrees, serves the purpose well. The depth of the blade and shaft guards against making the cut too deep. This modified cystotome (fig. 1) may also be used to make a scratch or ab externo incision or to outline the sclera to be removed in scleral resections.

Cystotome—Made by E. B. Meyrowitz Surgical Instrument Co., Inc., New York.

Presented as a *New Instrument* at the Fifty-Seventh Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 12-17, 1952, Chicago, Ill.

In Memoriam

Percy L. Balentine.....Philadelphia, Pennsylvania
December 12, 1951

S. Judd Beach.....Portland, Maine
February 10, 1953

Solomon Jefferson Chapman...Colorado Springs, Colorado
December 29, 1952

Elijah Rockhold Crossley.....Chicago, Illinois

Clarence E. Earnest.....Pueblo, Colorado
November 16, 1952

Goodlatte Browne Gilmore.....New York, New York
November 19, 1952

Roy Hampton Gough.....Fort Worth, Texas
December 1952

Howard Miller Hebble.....Moorestown, New Jersey
November 21, 1952

Frederick John Hoitash.....Huntington, West Virginia
November 29, 1952

Elgin Wilbur Jones.....Wakefield, Massachusetts
August 22, 1952

Carl Leonard Larson.....St. Paul, Minnesota
January 29, 1953

Louis Levy.....Memphis, Tennessee
October 20, 1952

John Alexander MacMillan.....Montreal, Quebec, Canada
February 6, 1953

John Silliman Macnie.....Minneapolis, Minnesota
January 30, 1953

Richard Edward Mercer.....Detroit, Michigan
October 28, 1952

Hugh Miller.....Kansas City, Missouri
September 11, 1952

Olin Winn Moss.....Lake Charles, Louisiana
January 9, 1953

Elizabeth Rindlaub.....Fargo, North Dakota
August 5, 1952

William Aaron Stoutenborough.....Columbus, Ohio
January 19, 1953

Eugene Emerson Willison.....Merced, California
January 24, 1953

TRANSACTIONS
American Academy of Ophthalmology and Otolaryngology
Official Publication of the American Academy of Ophthalmology and Otolaryngology
Published under the direction of the Editorial Board
WILLIAM L. BENEDICT, Rochester, Minnesota, *Editor*
W. HOWARD MORRISON, Omaha, Nebraska, *Associate Editor*
HENRY L. WILLIAMS, Rochester, Minnesota, *Associate Editor*
Printed by Douglas Printing Company, 109 North 18th Street, Omaha 2, Nebraska

All communications regarding the TRANSACTIONS should be addressed to William L. Benedict, M.D., Executive Secretary-Treasurer, American Academy of Ophthalmology and Otolaryngology, 100 First Avenue Building, Rochester, Minnesota

AMERICAN ASSOCIATION OF EYE, EAR, NOSE AND THROAT SOCIETIES SECRETARIES ANNUAL MEETING — 1952

The thirteenth annual meeting of the American Association of Eye, Ear, Nose and Throat Societies Secretaries was held at the Palmer House, Chicago, Illinois, at six o'clock, Tuesday, October 14, 1952, during the convention of the American Academy of Ophthalmology and Otolaryngology. The Academy again extended its courtesy to this secretarial group by providing an excellent dinner for the members and their guests.

The meeting was called to order by the President, Dr. Moacyr E. Alvaro of Sao Paulo, Brazil. Dr. Alvaro reminded the members that the dinner was provided by the American Academy of Ophthalmology and Otolaryngology and he thanked the officers of the Academy for their interest in the welfare and activities of the Secretaries' organization. He welcomed the 48 Secretaries who were present, including three from South America and one from Canada. Dr. Alvaro then presented Dr. A. D. Ruedemann, Secretary of Instruction for Ophthalmology, who spoke of some of the problems concerned with the annual meeting of the American Academy of Ophthalmology and Otolaryngology. He stressed the importance of a program which would interest all types of members, the older as well as the younger groups. He described the difficulty of getting new and interesting subjects for the study courses. He praised the work of our Secretaries, who have responded so well by writing to Dr. William L. Benedict, Executive Secretary-Treasurer of the American Academy of Ophthalmology and Otolaryngology, informing him of any capable physician who would be willing to present a scientific paper or give a study course which would be of interest to the members of the Academy. He urged that each local society secretary continue to write these constructive letters to Dr. Benedict.

Dr. Alvaro then reported on the progress of the work of our Society in extending the purpose of our group to include all the nations of the world. He reported that many societies all over the world had written to him expressing their desire to take an active part in an international group of eye, ear, nose and throat society secretaries. They expressed the feeling that a great amount

of international good will would be engendered by the work of such a society together with the many benefits that each local society would derive from knowing what other societies are doing to promote better educational meetings among eye, ear, nose and throat societies.

It was moved, seconded and unanimously passed that the name of our organization be changed to International Association of Eye, Ear, Nose and Throat Society Secretaries. It is hoped that all of our members will attempt to correspond with the secretaries of eye, ear, nose and throat societies all over the world, exchanging program announcements and other suggestions on how to develop a strong local society.

The remaining program consisted of a panel discussion on the subject, "The Selection of Good Speakers and Interesting Programs." Members of the panel were Dr. Paul C. Craig, Secretary for Instruction, Pennsylvania Academy of Ophthalmology and Otolaryngology; Dr. Sam H. Sanders, Secretary, Section on Otolaryngology, American Medical Association; Dr. Kenneth L. Craft, Founder of the American Association of Eye, Ear, Nose and Throat Society Secretaries. Dr. D. S. DeStio was the moderator.

Dr. Sam Sanders, Memphis, Tennessee, Chairman of the Nominating Committee, gave the report of the Committee nominating the following: Dr. James Monahan, Shenandoah, Pennsylvania, President; Dr. Edley Jones, Vicksburg, Mississippi, President-Elect; Dr. Daniel S. DeStio, Pittsburgh, Pennsylvania, Secretary-Treasurer. Since there were no further nominations from the floor, all the nominees were unanimously elected.

Dr. Moacyr Alvaro escorted the new President of the Association, Dr. James Monahan, to the chair. Dr. Monahan closed the meeting by stressing two points to the Secretary-members. First, each local Society Secretary should write to Dr. William L. Benedict, Executive Secretary-Treasurer of the American Academy of Ophthalmology and Otolaryngology, making any suggestions that he desires concerning the American Academy's annual program. Second, all the Secretary-members should get behind the new project of making this new International Society function successfully in order to develop international good will.

D. S. DeStio, M.D.
Secretary-Treasurer

News - - - Notes

AMERICAN ORTHOPTIC COUNCIL

The annual examination of orthoptic technicians by the American Orthoptic Council will be conducted in August and October 1953.

The written examination will be non-assembled and will take place on Thursday, August 27, in certain designated cities and offices and will be proctored by designated ophthalmologists.

The oral and practical examinations will be on Saturday, October 10, in Chicago just preceding the meeting of the American Academy of Ophthalmology and Otolaryngology.

Application for examination will be received by the office of the Secretary of the American Orthoptic Council, Dr. Frank D. Costenbader, 1605 22nd St. N.W., Washington 8, D. C., and must be accompanied by the examination fee of $20.00. Applications will not be accepted after July 1, 1953.

The basic course in orthoptics sponsored by the American Orthoptic Council will be given in 1953 in the Department of Ophthalmology, College of Medicine, State University of Iowa, Iowa City, Iowa.

The course will consist of didactic lectures and practical demonstrations, given by outstanding teachers. It will be held from June 15, 1953, through August 15, 1953.

High school graduates are eligible to take this course, but preference will be given to applicants with at least two years of college training, or its equivalent.

The tuition is $150.00; $50.00 payable on application, the remaining $100.00 to be paid at the time of registration. Arrangements have been made for the participants to be housed in the Nurses Home and to have their board at the University Hospitals. The cost for room, board and laundry will be approximately $75.00 per month per person.

In order to be eligible to take the examination leading to certification by the American Orthoptic Council, a candidate must have, in addition to the basic course sponsored by the Council, or an equivalent course, a minimum of seven months of practical training under a certified orthoptic technician in an office or institution approved by the Council. The Council makes every effort to find suitable positions for training for all participants in the course who desire them.

For further information about the course or to apply for admission write to Hermann M. Burian, M.D., Chairman, Committee on Instruction, University Hospitals, Iowa City, Iowa.

AMERICAN SOCIETY OF PLASTIC AND RECONSTRUCTIVE SURGERY

The Foundation of the American Society of Plastic and Reconstructive Surgery announces the regulations of the Fourth Annual Essay Contest:

1. The contest is restricted to residents in training, and plastic surgeons who have been in practice no longer than five years.
2. Two main prizes are offered, each consisting of a 6 months plastic surgical scholarship with active assistantship and full maintenance in a number of selected leading services.
3. The subject matter of the essay must be the result of some original, either clinical or laboratory research in Plastic and Reconstructive Surgery and about 5,000 words in length.
4. Traveling expenses between the services in the United States will be allotted.
5. A Silver Plaque or Certificate of Honorable Mention is also offered for a winning essay in Senior Classification to contestants active in practice of Plastic and Reconstructive Surgery for more than five years.
6. Manuscripts will not be accepted by the Award Committee after August 1, 1953.

The winning essays will appear on the program of the Annual Meeting of the Society, to be held at Coronado, California, November 2-6, 1953.

The 1952 Scholarship Awards went to Dr. John Frederick North, Oxford, England, who wrote on "The Use of Preserved Heterografts of Cartilage (Bovine) in Plastic Surgery"; Dr. J. William Littler, New York, whose subject was "The

Neurovascular Pedicle Method of Digital Transposition for Subtotal Reconstruction of the Thumb"; Dr. Raymond O. Brauer, Houston, Texas, for his paper on "A Consideration of the LeMesurier Technic of Single Harelip Repair with a New Concept as to Its Use in Incomplete and Secondary Harelip Repair"; Dr. Sten Stenstrom, Stockholm, Sweden, who described "A New Form of Direct Flap Procedure"; and Dr. C. R. McLaughlin, East Grinstead, Sussex, England, for his discussion of "Surgical Support in Permanent Facial Paralysis."

For further information write to the Award Committee, c/o Jacques W. Maliniac, Chairman, 30 Central Park South, New York 19, N. Y.

LANCASTER COURSES IN OPHTHALMOLOGY

The Ophthalmic Study Council, 243 Charles Street, Boston, Massachusetts, will sponsor the Lancaster Courses in Ophthalmology, June 22 through September 5, 1953, at Colby College, Waterville, Maine. The usual excellent faculty will be on hand, and the courses given in former years will be repeated.

NATIONAL COUNCIL TO COMBAT BLINDNESS, INC.

Applications for the 1953-1954 grant-in-aid awards of the National Council to Combat Blindness will be considered by the organization's Committee on Research at its fourth annual meeting to be held in the Spring of 1953. Completed applications should be received in the office of the Council no later than April 15, 1953. Forms may be obtained by addressing: Secretary, National Council to Combat Blindness, Inc., 1186 Broadway, New York 1, N. Y.

DISTRICT OF COLUMBIA

A Spring meeting on the recent advances in Ophthalmology will be held at Walter Reed Army Medical Center on May 21 and 22, 1953. Speakers will include Professor A. Franceschetti of Geneva, Switzerland; Drs. Alston Callahan, Michael J. Hogan, Irving H. Leopold, and Frank B. Walsh; Mrs. Helenor Campbell Wilder; and Colonels H. Amory, J. H. King, Jr., and Austin Lowrey.

Interested civilian physicians are invited to attend. There is no fee. Application should be made by April 21, 1953, to Commanding General, Walter Reed Army Medical Center, Washington 12, D. C., Attention Colonel J. H. King, Jr., Director of the Ophthalmology Course.

ILLINOIS

The Chicago Ophthalmological Society held its 1953 Clinical Conference on February 12, 13 and 14.

Dr. Frank B. Walsh, Associate Professor of Ophthalmology at the Johns Hopkins University, gave the Ninth Annual Sanford Robinson Gifford Memorial Lecture. Dr. Walsh spoke on "Some Aspects of Calcification in Ophthalmology."

MICHIGAN

The Department of Postgraduate Medicine of the University of Michigan Medical School announces the annual conference in Ophthalmology for qualified physicians, April 20 to 22, 1953, to be given at the Horace H. Rackham Graduate School Building, Ann Arbor, under the direction of the Department of Ophthalmology at the University of Michigan Medical School.

Guest lecturers will be Dr. F. Phinizy Calhoun, Jr., Atlanta, Ga.; Dr. Isadore Givner, New York; Dr. David O. Harrington, San Francisco; Dr. M. Noel Stow, Washington, D. C.; and Dr. Walter Z. Rundles, Jr., Flint, Mich. Resident lecturers will include Dr. Harold F. Falls, Dr. William H. Havener, Dr. John Woodworth Henderson, Dr. Harry B. McGee, Dr. Stanley P. Oleksy, and Dr. James R. Quinn.

More complete information may be obtained by writing to Dr. Howard H. Cummings, Chairman, Department of Postgraduate Medicine, University Hospital, Ann Arbor, Michigan.

NEW YORK

The Alumni Association of the New York Eye and Ear Infirmary will hold its annual scientific program April 20 through April 24. The scientific sessions in Ophthalmology will be held Monday and Tuesday. The ear, nose and throat on Thursday and Friday at the New York Academy of Medicine in room 440. Morning sessions will commence at nine-thirty, afternoon sessions at two. Wednesday will be left open for clinical and surgical demonstrations at the New York Eye and Ear Infirmary.

Dr. Julius Lempert, Director of the Lempert Institute of Otology, New York, will be guest-participant in panel discus-

sions at the First Western Hemisphere Conference of the World Medical Association at Richmond, Va., on April 24, 1953.

The sessions will provide opportunity for a discussion of current medical problems between representatives of the national medical societies of Latin America and United States specialists and practitioners. Dr. Louis H. Bauer, president of the American Medical Association and secretary-general of the World Medical Association, will moderate at a general session at which panel reports will be made.

Dr. Lempert will contribute a chapter on major advances in the field of otolaryngology to a volume commemorating the conference. He is a diplomate of the American Board of Otolaryngology and is a member of the American Academy of Ophthalmology and Otolaryngology.

A course in glaucoma with particular emphasis on gonioscopy and study of the anterior angle will be given at the Brooklyn Eye and Ear Hospital on May 4, 5 and 6, 1953. Ample opportunity for practical instruction in the use of the gonio-lens will be given and material from the Glaucoma Clinic will be utilized. One afternoon will be devoted to surgical indications and technic of glaucoma surgery in the operating room.

On the faculty will be Dr. E. Clifford Place, Dr. Walter V. Moore, Dr. M. A. Lasky, Dr. Daniel Kravitz, and Dr. Arthur Shainhouse.

Application and fee of $40.00 for registration by ophthalmologists may be addressed to Dr. Daniel Kravitz, Brooklyn Eye and Ear Hospital, 29 Greene Avenue, Brooklyn 38, New York.

NORTH CAROLINA

On November 24, 1952, the Charlotte Ophthalmological Society was formed by the following eye physicians: C. B. Foster, W. R. Graham, T. D. Ghent, H. T. Holden, M. J. Hough, Ruth Leonard, M. J. Lymberis, H. C. Neblett, F. C. Smith and J. D. Stratton. The officers elected were Dr. H. C. Neblett, president, Dr. J. D. Stratton, vice-president, and Dr. T. D. Ghent, secretary and treasurer. Meetings will be held bimonthly and will be of a scientific nature.

OREGON

The Oregon Academy of Ophthalmology and Otolaryngology announces the Annual Spring Convention in Ophthalmology and Otolaryngology to be held in Portland, March 23 to 27, 1953. An excellent program has been arranged by the Oregon Academy and the University of Oregon Medical School. Ophthalmology sessions are scheduled on March 23, 24 and the morning of March 25. Otolaryngology sessions will be held the afternoon of March 25, and on March 26 and 27.

Guest speakers will be Dr. Hermann Burian, Associate Professor of Ophthalmology, State University of Iowa Medical School, Iowa City; Dr. Harold Scheie, Associate Professor of Ophthalmology, University of Pennsylvania Medical School, Philadelphia; Dr. Kenneth M. Day, Professor of Otology, University of Pittsburgh Medical School, Pittsburgh; and Dr. Jerome A. Hilger, Clinical Assistant Professor of Otolaryngology, University of Minnesota Medical School, Minneapolis.

Dr. Burian will deliver the fourth John E. Weeks Memorial Lecture in Ophthalmology.

PENNSYLVANIA

The Section on Ophthalmology of the College of Physicians of Philadelphia met on Thursday, January 15, 1953. Dr. W. Morton Grant, Boston, Mass., the guest speaker, spoke on "Open Angle Glaucoma."

Dr. Patrick J. Kennedy has received the appointment as an Attending Surgeon at the Wills Eye Hospital succeeding Dr. William J. Harrison, who served in that capacity from 1939. Dr. Harrison will now serve as a Consulting Surgeon to the hospital.

SOUTH CAROLINA

A joint meeting of the South Carolina Society of Ophthalmology and Otolaryngology with the North Carolina Eye, Ear, Nose and Throat Society will be held on September 14, 15, and 16, 1953. Headquarters will be the Francis Marion Hotel, Charleston, S. C.

The following otolaryngologists will be on the program; Dr. Wayne Slaughter and Dr. John R. Lindsay of Chicago, and Dr. Theodore Walsh of St. Louis. Ophthalmological subjects will be covered by Dr. Derrick Vail of Chicago, Dr. David G. Cogan of Boston, and Dr. R. Townley Paton of New York.

There will be a panel on vertigo to be discussed from the ophthalmological and

otolaryngological, neurological, and internal medical standpoints.

For further information please communicate with Dr. George T. Ferguson, secretary of the North Carolina Eye, Ear, Nose and Throat Society, McPherson Hospital, Durham, N. C., or Dr. Roderick Macdonald, Secretary of the South Carolina Society of Ophthalmology and Otolaryngology, 330 East Main Street, Rock Hill, S. C.

VIRGINIA

The Gill Memorial Eye, Ear and Throat Hospital will present its Twenty-Sixth Annual Spring Congress in Ophthalmology, Otolaryngology and allied specialties on April 6 to 11, 1953 at Roanoke, Va.

Guest lecturers will be Dr. A. B. Reese, Clinical Professor of Ophthalmology, College of Physicians and Surgeons, Columbia University and Surgeon and Pathologist to the Institute of Ophthalmology, New York; Dr. Louis H. Bauer, President of the American Medical Association, Hempstead, New York; Dr. H. B. Stallard, Surgeon, Moorfields Eye Hospital, and Ophthalmic Surgeon, St. Bartholomew's Hospital, London, England; Major General George E. Armstrong, Surgeon General of the United States Army, Washington, D. C. and Dr. Wallace E. Herrell, Professor of Medicine, Mayo Foundation Graduate School of Medicine of the University of Minnesota, and Head of a Section of Medicine, Mayo Clinic, Rochester, Minn.

GERMANY

The annual meeting of the German Society for Otorhinolaryngology will be held in Wiesbaden May 19 to 23. The following topics have been scheduled for discussion: "Esophagoscopy and Endoscopic Treatment of the Esophagus," "Surgery of the Esophagus," and "Relations of the Sympathetic Nervous System to Otorhinolaryngologic Diseases."

NEW ZEALAND

The Annual Meeting of the Ophthalmological Society of New Zealand for the year 1952 was held in Auckland during September. The members of the Society were greatly honored by the presence of Dr. Algernon Reese of New York and Mr. John Foster of Leeds, Great Britain. Both these gentlemen read papers and took a very active part in the deliberations of the Society. The next annual meeting of the Ophthalmological Society of New Zealand will probably take place in Dunedin during October 1953.

POSITIONS AVAILABLE

The American Association of Orthoptic Technicians wishes to announce that a Confidential Placement Service has been organized for positions for Certified Orthoptic Technicians. Information regarding available technicians and positions open can be received by writing Mrs. Louisa Wells Kramer, 1779 Massachusetts Avenue N.W., Washington 6, D. C.

We are in need of a resident in Ophthalmology, to begin May 1 or July 1; also, a vacancy exists in our Department of Otolaryngology, now or July 1. Each is a two-year residency. If further information is desired, contact the Medical Director of the University of Oklahoma Hospitals, 800 Northeast Thirteenth St., Oklahoma City 4, Oklahoma.

Assistant residency in ophthalmology will be available July 1, 1953, as a result of unexpected demands of the military. This service is a medical school service, fully approved by the American Medical Association, with very active out-patient service and busy surgical service. Usual resident's stipend and up-keep may be supplemented by participation in active research program now underway. Training in basic sciences or previous experience in ophthalmology desirable but not absolutely necessary. Write Dr. Winston Roberts, Chief of Ophthalmology, Bowman Gray School of Medicine and North Carolina Baptist Hospital, Winston-Salem, N. C.

Listings of positions available should be sent to W. L. Benedict, M.D., 100 First Avenue Building, Rochester, Minn., by the fifth of the month preceding publication. They should include (1) type of physician wanted (ophthalmologist or otolaryngologist; diplomate, resident, etc.); (2) type of position to be filled; and (3) whom to write for further information. Unless otherwise requested, listings will be published once only.

The Academy will handle no further correspondence beyond the listing and assume no responsibility. Neither does it endorse nor guarantee any of the published listings.

DIRECTORY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC SOCIETIES

INTERNATIONAL

International Association for Prevention of Blindness

President: Dr. P. Bailliart, 47 Rue de Bellechasse, Paris, France
Secretary-General: Prof. A. Franceschetti, rue A—Jentzer 22, Geneva, Switzerland

International Broncho-Esophagological Society

President: Dr. Fernand Eeman
Secretary: Dr. Chevalier L. Jackson, 1901 Walnut Street, Philadelphia 3, Pa.
Time and Place: Second International Congress of Bronchoesophagology, June 15 and 16, 1953, Albert Plage, Le Zoute, Belgium.

International Council of Ophthalmology

President: Sir Stewart Duke-Elder
Secretary: Prof. Dr. Edward Hartmann, 2, Avenue Ingres, Paris (16e) France

International Organization Against Trachoma

President: Dr. A. F. MacCallan
Secretary: Dr. F. Wibaut, P. C. Hoofstraat 145, Amsterdam, Holland

Pan-American Association of Ophthalmology

President: Dr. M. E. Alvaro
Secretary: Dr. Thomas D. Allen, 122 South Michigan Avenue, Chicago 2, Ill.
Time and Place: February 1956. Santiago, Chile

Pan-American Association of Ophthalmology, Puerto Rico Chapter

President: Dr. Luis J. Fernandez, Box 2206, San Juan 10, Puerto Rico
Secretary: Dr. P. Fernandez
Place: San Juan

Pan-American Association of Oto-Rhino-Laryngology and Broncho-Esophagology

President: Dr. Justo M. Alonso
Secretary: Dr. Chevalier L. Jackson, 1901 Walnut Street, Philadelphia 3, Pa.
Time and Place: Fourth Pan-American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology, February 28 to March 4, 1954, Mexico City

FOREIGN

The Ophthalmological and Otolaryngological Society of Alberta

President: Dr. W. E. Spankie
Secretary: Dr. M. R. Marshall, Wells Pavilion, University Hospital, Edmonton, Alberta, Canada
Time and Place: Spring of 1953 in Edmonton

All-India Ophthalmological Society

President: Dr. G. Zachariah
Secretaries: Dr. S. N. Cooper and Dr. V. K. Chitnis, Laud Mansion, Queen's Road, Bombay 4, India

Associacao Medica do Instituto Penido Burnier

President: Dr. Leoncio de Souza ueiroz
First Secretary: Dr. Milton B. de Toledo
Second Secretary: Dr. Aloisio Af. Ferreira

Austrian Otolaryngological Society, Vienna

President: Prof. Dr. E. Schlander
Secretary: Doz. Dr. O. Novotny, Vienna IX, Alserstrasse 4, Austria
Time: Annually

Belgian Ophthalmologic Society

President: Dr. L. Weekers
Secretary-General: Dr. M. Appelmans, Avenue Ruelens 179, Louvain
Time and Place: January, June and November. Bruxelles

Bombay Ophthalmologists' Association

Chairman: Rotated
Conveners: Dr. S. N. Cooper and Dr. B. D. Telang, Laud Mansion 21, Queen's Road, Bombay 4
Time and Place: 7:30 p.m., third Wednesday of each month. The seven ophthalmic hospitals of Bombay by rotation

British Association of Otolaryngologists

President: Mr. V. E. Negus
Honorary Secretary: Mr. F. C. W. Capps, 45 Lincoln's Inn Fields, London W. C. 2, England

British Medical Association, Section on Ophthalmology

President: Mr. O. G. Morgan
Secretary: Mr. A. G. Cross, 27 Harley Street, London W. 1, England

Centro de Estudos de Oftalmologia de Sao Paulo

President: Dr. Rubens Belfort Mattos
Secretary: Dr. Jose Carlos Gouvea Pacheco, Marconi 34, Sao Paulo

Chengtu Ophthalmological Society

President: Dr. Eugene Chan
Secretary: Dr. D. S. Shen, Eye, Ear, Nose and Throat Hospital, Chengtu, Szechuan, China
Place: Eye, Ear, Nose and Throat Hospital, Chengtu, Szechuan, China

Chinese Ophthalmology Society

President: Dr. C. H. Chou
Secretary: Dr. F. S. Tsang, 221 Foochow Road, Shanghai

Chinese Ophthalmological Society of Peking

President: Dr. G. C. Lin
Secretary: Dr. H. L. Chen, Ophthalmological Dept., Peking University Medical School, Peking
Time: Bimonthly

Deutsche Ophthalmologische Gesellschaft Heidelberg

President: Prof. Dr. med. K. Wessely
Secretary: Prof. Dr. med. E. Engelking, Heidelberg, Universitäts-Augenklinik

Faculty of Ophthalmologists

President: Mr. O. M. Duthie
Secretary: Mr. J. H. Doggart, F.R.C.S., 45 Lincoln's Inn Fields, London, W.C. 2, England

Secretaries of societies are requested to furnish the information necessary to make this list complete and keep it up to date.

FRENCH SOCIETY OF OPHTHALMOLOGY
Secretary-General: Dr. Marcel Kalt, 81 Rue St. Lazare, Paris 9, France
Time and Place: June 20, 1953 and May 16, 1954, Paris

FRENCH SOCIETY OF OTOLARYNGOLOGY
Secretary: Dr. H. Flurin, 19 Rue MacMahon, Paris, France

GERMAN OPHTHALMOLOGICAL SOCIETY
President: Prof. W. Lohlein
Secretary: Prof. E. Engelking, Heidelberg

HUNGARIAN MEDICAL TRADE UNION, SECTION OF OPHTHALMOLOGY
President: Prof. I. Csapody
Secretary: Dr. E. Galla, Krisztina Körut 139, Budapest, Hungary
Time and Place: Bimonthly. Second Eye Clinic, Budapest

HUNGARIAN OPHTHALMOLOGICAL SOCIETY
President: Prof. G. Horay
Assistant Secretary: Dr. Stephen de Grosz, University Eye Hospital, No. 1, Illesucca 15. Budapest

ISRAEL OPHTHALMOLOGICAL SOCIETY
President: Dr. Aryeh Feigenbaum
Secretary: Dr. E. Sinai, 9 Bialik Street, Tel Aviv

MIDLAND OPHTHALMOLOGICAL SOCIETY
President: Dr. F. A. Anderson
Secretaries: Dr. P. Jameson Evans, 51 Calthorpe Road, Edgbaston, Birmingham 15, England
Dr. R. D. Weeden Butler, 18 Highfield Road, Edgbaston, Birmingham 15, England
Place: Birmingham and Midland Eye Hospital, Church Street, Birmingham 3, England

NEDERLANDSCH OOGHEELKUNDIG GEZELSCHAP
President: Prof. Dr. H. M. Dekking
Secretary: Dr. T. A. Vos, Laan v. Meerdervoort 394, Gravenhage

NORTH OF ENGLAND OPHTHALMOLOGICAL SOCIETY
President: Mr. A. McKie Reid
Secretary: Mr. W. M. Muirhead, 70 Upper Hanover Street, Sheffield 3, England
Time and Place: October to May. Manchester, Leeds, Newcastle-on-Tyne, Liverpool, Sheffield and Bradford

SOCIETY OF NORWEGIAN OPHTHALMOLOGISTS
President: Dr. J. C. Holst, Pilestredet 28, Oslo
Secretary: Dr. Thore Lie Thomassen
Place: Rikshospitalets Oeyenklinikk, Oslo

THE NOVA SCOTIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. C. K. Fuller
Secretary-Treasurer: Dr. E. I. Glenister, 513 Barrington Street, Halifax, Nova Scotia
Time: Four times yearly at dates to be arranged by the Executive

OPHTHALMOLOGICAL SOCIETY OF AUSTRALIA
President: Dr. A. L. Tostevin
Secretary: Dr. Arnold L. Lance, 27 Commonwealth Street, Sydney, New South Wales
Time and Place: October 12, 1953, Adelaide, South Australia

OPHTHALMOLOGICAL SOCIETY OF COPENHAGEN
President: Prof. Dr. Victor Larsen
Secretary: Dr. S. Ry Andersen, Rigshospitalets oejenafdeling, Copenhagen Oe, Denmark
Place: Rigshospitalets oejenafdeling

OPHTHALMOLOGICAL SOCIETY OF EGYPT
President: Dr. Ibrahim Ahmad Mohammad
Secretary: Dr. Mohammad Khalil, 15 A Sherif Pasha, Str., Cairo
Time and Place: Annual meeting during March. Summer meeting during August. Dar El Hekmah 42 Kasr El Ainy Street, Cairo, Egypt

OPHTHALMOLOGICAL SOCIETY OF HOSPITAL DE NUESTRA SENORA DE LA LUZ
Chairman: Dr. Manuel J. Icaza y Dublan
Secretary: Dr. Jorge Meyran, Ezequiel Montes 135, México, D. F.
Time and Place: Second Friday of each month. Hospital de Nuestra Senora de la Luz

OPHTHALMOLOGICAL SOCIETY OF NEW ZEALAND
President: Dr. Howard Coverdale
Secretary: Dr. W. J. Hope-Robertson, Kelvin Chambers, 16 The Terrace, Wellington
Time and Place: Annually. Auckland, Wellington, Christchurch and Dunedin alternately

OPHTHALMOLOGICAL SOCIETY OF SOUTH AFRICA
President: Dr. L. Staz
Secretary-Treasurer: Dr. J. Guillaume Louw, 901 Dumbarton House, Adderley Street, Cape Town
Time: Annually

OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM
President: Dr. A. MacRae
Honorary Secretaries: Mr. A. G. Leigh and Mr. Harold Ridley, 45 Lincoln's Inn Fields, London
Time and Place: April 23, 24, and 25, 1953, London

OPHTHALMOLOGISCHE GESELLSCHAFT IN WIEN
President: K. Lindner
Secretary: Ch. Eyb, Wien, IX, Alserstrasse 4

OXFORD OPHTHALMOLOGICAL CONGRESS
Master: Dr. F. A. Anderson
Hon. Secretary and Treasurer: Ian C. Fraser, F.R.C.S., Red Roofs, Kingsland, Shrewsbury

PHILIPPINE OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY
President: Dr. Jesus Eusebio
Secretary-Treasurer: Dr. Jesus Tamesis, Philippine General Hospital, Manila

POLISH OPHTHALMOLOGICAL SOCIETY
President: Prof. Dr. W. Kapuscinski
Secretary: Dr. S. Topolski, Piusa 38, Warsaw, Poland
Time and Place: Every two years—summer. Ophthalmic Clinic, Oczki 6, Warsaw

ROYAL SOCIETY OF MEDICINE, SECTION OF OPHTHALMOLOGY
President: Montague Hine, F.R.C.S.
Secretaries: Arthur Lister, F.R.C.S., 56 Wimpole Street, London, W.1., England
H. E. Hobbs, F.R.C.S., 129 Harley Street, London, W.1., England

SAO PAULO SOCIETY OF OPHTHALMOLOGY

President: Dr. Durval Prado
Secretary: Dr. Rubens Belfort Mattos, Rua B. Stapetininga, 29 F-3° andar, Sao Paulo, Brazil

SCOTTISH OPHTHALMOLOGICAL CLUB

President: Dr. E. H. Cameron
Secretary: Dr. John Marshall, 11 Clairmont Gardens, Glasgow, C.3, Scotland
Time and Place: Last Saturday of March and October, Edinburgh and Glasgow, in rotation

SOCIEDAD ARGENTINA DE OFTALMOLOGIA

President: Dr. Jorge Malbran
Secretary: Dr. Jose Maria Roveda, Lezica 4472, Buenos Aires
Time and Place: Third Wednesday of every month. Buenos Aires

SOCIEDAD CHILENA DE OFTALMOLOGIA

President: Dr. Italo Martini
Secretary: Dr. Adrian Araya Costa, Renaca, no. 34, Santiago, Chile

SOCIEDAD COLOMBIANA DE OFTALMOLOGIA Y DE OTORHINOLARINGOLOGIA

President: Dr. Jorge Suarez-Hoyos, Carrera 5a, no. 13-39, Bogota, Colombia
Secretary: Dr. Francisco Arango
Time and Place: Second Tuesday of each month. Club Medico

SOCIEDAD CUBANA DE OFTALMOLOGIA

President: Dr. Gilberto Cepero Garcia
Secretary: Dr. Jose Alfonso, Jr.
Time and Place: First Thursday bi-monthly. Malecon No. 61 Bajos, Havana, Cuba

SOCIEDAD MEXICANA DE OFTALMOLOGIA

President: Dr. Teodulo M. Agundis
Secretary: Dr. Jose Luis Arce, Viena 3-5, Mexico D. F., Mexico
Time and Place: 8:30 p.m., first Tuesday of each month, Gral. PRIM 47

SOCIEDAD NICARAGUENSE DE OFTALMOLOGIA

President: Dr. Ricardo Lacayo
Secretary: Dr. Gilberto Cuadra, Clinica Especializada, Managua, D. N., Nicaragua, C. A.

SOCIEDAD DE OFTALMOLOGIA DE CORDOBA

Chairman: Dante J. Yadarola
Secretary: Dr. Alberto Urrets Zavalia (hijo), 27 de Abril 255, Cordoba, Argentina

SOCIEDAD DE OFTALMOLOGIA DE GUADALAJARA

President: Dr. Elias Mendoza Gonzalez
Secretary: Dr. Jose Martin del Campo, Av. Juarez 211, Desp. 314, Guadalajara, Jal.
Place: Edificio Lutencia Despacho 101

SOCIEDAD OFTALMOLOGICA HISPANO AMERICANA

President: Dr. Soria
Secretary: Dr. Costi, 3. Calle Montalban, Madrid

SOCIEDAD DE OFTALMOLOGIA DEL LITORAL

President: Dr. Juan Manuel Vila Ortiz, Cordoba 1915, Rosario, Argentina
Secretary: Dr. Carlos M. Soto
Time and Place: Last Sunday in every month. Rosario

SOCIEDAD OFTALMOLOGICA DE VALPARAISO

President: Prof. Dr. Jean H. Thierry
Secretary-Treasurer: Dr. Jorge Oyarzun O, Hospital de Ojos, Calle Blas, Cuevas, Valparaiso

SOCIEDAD PERUANA DE OTO-RINO-LARINGOLOGIA Y OFTALMOLOGIA

President: Dr. Enrique Cipriani V., Pasaje Olaya 156, Lima, Peru
Secretario General: Dr. Enrique Haro
Secretario de Actas: Dr. Alberto Castillo R.

SOCIEDADE BRASILEIRA DE OFTALMOLOGIA

President: Dr. Paulo C. Pimental
First Secretary: Dr. Pedro Moacyr de Aguiar, Ladeira Santa Thereza 136, Apt. 101, Rio de Janeiro
Second Secretary: Dr. Luiz Enrico Ferreira
Time: Third Friday of every month from April to December

SOCIEDADE DE OFTALMOLOGIA DE MINAS GERAES

President: Dr. Amelio Bonfioli
Secretary: Dr. Mariana Noronha, Pauso Alegre 1150, Belo Horizonte, Est. Minas Geraes

SOCIEDADE DE OFTALMOLOGIA DEL NORTE

President: Dr. Jorge Luis Castillo
Secretary, Dr. Felix Berman, San Lorenzo 345, Tucuman, Argentina
Place: Mendoza 421, Tucuman

SOCIEDADE DE OFTALMOLOGIA E OTO-RHINO-LARYNGOLOGIA DE BAHIA

President: Dr. Theonilo Amorim, Barra Avenida, Bahia, Brazil
Secretary: Dr. Adroaldo de Alencar

SOCIEDADE DE OFTALMOLOGIA E OTO-RINO-LARINGOLOGIA DO RIO GRANDE DO SUL

President: Dr. Mario Araujo Azambuja
First Secretary: Dr. Humberto Lubisco, Rua Donna Laura 45, Porto Alegre, Est., Rio Grande do Sul
Second Secretary: Dr. Ivo Kuhl

SOCIEDADE DE OFTALMOLOGIA DE SAO PAULO

President: Dr. Paulo Braga Magalhaes
General Secretary: Dr. Paulo Aranha de Azevedo, Xavier de Toledo 71,1° and., Sao Paulo
First Secretary: Dr. Alfredo Rocco

SOCIEDADE DE OTO-RINO-LARINGOLOGIA DO RIO DE JANIERO

President: Dr. Aloysio Novis
Secretaries: Dr. Rubens Cabral, Rua Paissandu 73, Rio de Janeiro, Brazil
Dr. Mairelles Vieira

SOCIETA OFTALMOLOGICA ITALIANA

President: Prof. Giuseppe Ovio
Secretary: Prof. E. Leonardi, Piazza degli Eroi, 11 Roma, Italy
Place: Roma Piazza degli Eroi 11

SOCIETE BELGE D'OPHTALMOLOGIE

President: Dr. L. Weekers
Secretary-General: Dr. M. Appelmans, 179 avenue Reulens, Louvain, Belgium
Time: Last Sunday of February, June and November

SOCIETE FRANCAISE D'OTO-RHINO-LARYNGOLOGIE

Administrateur: G. Raimond, 17et19, Rue de Buci, Paris (VIe) France

SOCIETY OF SWEDISH OPHTHALMOLOGISTS

President: Prof. S. Larsson
Secretary: Dr. K. O. Granstrom, Sodermalmstorg 4, Stockholm So, Sweden
Time: Annual meeting, beginning of June

SOUTHERN OPHTHALMOLOGICAL SOCIETY, ENGLAND

President: Campbell Shaw, M. R. C. S.
Hon. Secretary and Treasurer: Nigel Cridland, D.M., D.O., 25 Craneswater Park, Southsea, England

NATIONAL

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Frederick C. Cordes
Executive Secretary-Treasurer: Dr. William L. Benedict, 100 First Avenue Building, Rochester, Minn.
Time and Place: October 11-16, 1953, Chicago

AMERICAN ASSOCIATION OF EYE, EAR, NOSE AND THROAT SOCIETY SECRETARIES

President: Dr. James J. Monahan
Secretary-Treasurer: Dr. Daniel S. DeStio, Highland Building, Pittsburgh 6, Pa.

AMERICAN LARYNGOLOGICAL ASSOCIATION

President: Dr. Louis H. Clerf
Secretary: Dr. Harry P. Schenck, 326 S. 19th Street, Philadelphia 3, Pa.
Time and Place: April 26-27, 1953, The Roosevelt, New Orleans, La.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President: Dr. Francis E. LeJeune
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Building, Rochester 7, N. Y.
Time and Place: April 28-30, 1953, The Roosevelt, New Orleans, La.

EASTERN SECTION
Vice-President: Dr. Irl H. Blaisdell
Secretary: Dr. Francis W. Davison, Geisinger Memorial Hospital, Danville, Pa.
Time and Place: January 7, 1953, Hotel Syracuse, Syracuse, N. Y.

MIDDLE SECTION
Vice-President: Dr. Henry L. Williams
Secretary: Dr. Walter H. Theobald, 307 North Michigan Avenue, Chicago 1, Ill.
Time and Place: January 19, 1953, The Drake, Chicago, Ill.

SOUTHERN SECTION:
Vice-President: Dr. W. W. Wilkerson, Jr.
Secretary: Dr. James W. McLaurin, Raymond Building, Baton Rouge 6, La.
Time and Place: January 12, 1953, Andrew Jackson Hotel, Nashville, Tenn.

WESTERN SECTION:
Vice-President: Dr. Alden H. Miller
Secretary: Dr. Harold Boyd, 1136 West Sixth Street, Los Angeles, Calif.
Time and Place: January 24, 1953, The Elks Club, Los Angeles, Calif.

AMERICAN MEDICAL ASSOCIATION, SCIENTIFIC ASSEMBLY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Edwin B. Dunphy
Secretary: Dr. Trygve Gundersen, 101 Bay State Road, Boston, Mass.

AMERICAN MEDICAL ASSOCIATION, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY

Chairman: Dr. James Milton Robb
Secretary: Dr. Sam H. Sanders, 1089 Madison Avenue, Memphis 3, Tenn.

AMERICAN OPHTHALMOLOGICAL SOCIETY

President: Dr. Conrad Berens
Secretary-Treasurer: Dr. Maynard C. Wheeler, 30 W. 59th Street, New York 19, N. Y.
Time and Place: May 28-30, 1953, The Homestead, Hot Springs, Va.

AMERICAN OTOLOGICAL SOCIETY

President: Dr. Albert C. Furstenberg
Secretary: Dr. John R. Lindsay, 950 East 59th Street, Chicago 37, Ill.
Time and Place: May 1-2, 1953, Roosevelt Hotel, New Orleans, La.

AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY

President: Dr. Norman N. Smith
Secretary: Dr. Joseph G. Gilbert, 1404 President Street, Brooklyn 13, N. Y.

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY

President: Dr. Kenneth L. Craft
Secretary-Treasurer: Dr. Michael H. Barone, 468 Delaware Ave., Buffalo, N. Y.
Time and Place: Annual meeting, October 16, 1953, Chicago

ASSOCIATION FOR RESEARCH IN OPHTHALMOLOGY, INC.

Chairman: Dr. Walter H. Fink
Secretary-Treasurer: Dr. James H. Allen, 1430 Tulane Avenue, New Orleans, La.

CANADIAN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY

President: Dr. R. G. C. Kelly
Secretary: Dr. J. Clement McCulloch, 380 Medical Arts Building, Toronto, Ont.

CANADIAN OPHTHALMOLOGICAL SOCIETY

President: Dr. Kenneth B. Johnston
Secretary: Dr. R. G. C. Kelly, 113 St. Clair Avenue W., Toronto 5, Ont., Canada

CANADIAN OTOLARYNGOLOGICAL SOCIETY

President: Dr. Jules Brahy
Secretary: Dr. W. Ross Wright, 361 Regent Street, Fredericton, New Brunswick

NATIONAL SOCIETY FOR THE PREVENTION OF BLINDNESS, INC.

President: Mr. Mason H. Bigelow
Secretary: Dr. Franklin M. Foote, 1790 Broadway, New York 19, N. Y.

SOCIETY OF MILITARY OPHTHALMOLOGISTS

President: Col. Austin Lowrey
Secretary-Treasurer: Col. J. H. King, Jr., Walter Reed Army Hospital, Washington 12, D. C.
Time and Place: October, 1953, Walter Reed Army Hospital, Washington 12, D. C.

REGIONAL

ARK-LA-TEX OTO-OPHTHALMIC SOCIETY

President: Dr. Joel A. Wilkinson
Secretary: Dr. Frank L. Bryant, 2622 Greenwood, Shreveport, La.
Time and Place: First Monday of month, September through June. Shreveport Club

HAWAII EYE, EAR, NOSE AND THROAT SOCIETY

Chairman: Dr. Clarence W. Trexler
Secretary: Dr. Tadao Hata, 704 S. King St., Honolulu
Time and Place: Third Thursday of each month, September through May (dinner meetings) 6:30 p.m., Pacific Club, Honolulu

INTER-MOUNTAIN OTO-OPHTHALMOLOGICAL SOCIETY

President: Dr. Robert G. Snow
Secretary-Treasurer: Dr. Whitney J. Haight, 1015 Boston Building, Salt Lake City 1, Utah
Time and Place: Third Monday of each month, September through May. University Club, 136 East South Temple, Salt Lake City

LOUISIANA-MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President Dr. W. L. Hughes
Secretary: Dr. Edley H. Jones, 1301 Washington Street, Vicksburg, Miss.
Time and Place: May 11, 1953, Edgewater Gulf Hotel, Edgewater, Miss.

NASSAU OPHTHALMOLOGICAL SOCIETY

President: Dr. A. Milton Goldman
Secretary: Dr. Ward L. Mould, 131 Fulton Avenue, Hempstead, N. Y.
Time and Place: Fourth Monday in February, April, September and November at 7:30 p.m. at Meadow brook Hospital, Hempstead, N. Y.

NEW ENGLAND OPHTHALMOLOGICAL SOCIETY

President: Dr. Benjamin A. Sachs
Secretary: Dr. Garrett E. Sullivan, 101 Bay State Road, Boston 15, Mass.
Time and Place: Third Wednesday of each month, November through April. Massachusetts Eye and Ear Infirmary

NEW ENGLAND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. Leighton F. Johnson
Secretary-Treasurer: Dr. Burton E. Lovesey, 76 Bay State Road, Boston 15, Mass.
Time and Place: Quarterly. Massachusetts Eye and Ear Infirmary, 243 Charles Street, Boston

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY

President: Dr. C. Allen Dickey
Secretary-Treasurer: Dr. Howard P. House, 1136 West Sixth Street, Los Angeles 14, Calif.
Time and Place: 1953, Los Angeles, Calif.

SAGINAW VALLEY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. William B. Hubbard
Secretary: Dr. Frank A. Ware, 514 Genesee Bank Bldg., Flint, Mich.
Time and Place: Second Tuesday evening of each month, except summer months. Bancroft Hotel, Saginaw

SIOUX VALLEY EYE AND EAR ACADEMY

President: Dr. James Reeder, Jr.
Secretary-Treasurer: Dr. W. P. Davey, 627-632 Frances Building, Sioux City, Iowa

SOUTHERN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. Edley H. Jones
Secretary: Dr. F. A. Holden, Medical Arts Building, Baltimore, Md.

TIDEWATER OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. C. C. Cooley
Secretary: Dr. J. G. Sellers, 413 Medical Arts Building, Norfolk, Va.

WISCONSIN-UPPER MICHIGAN SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. H. D. McEachran
Secretary: Dr. G. L. McCormick, 650 South Central Avenue, Marshfield, Wis.

STATE

ALABAMA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Ralph Clements
Secretary-Treasurer: Dr. David McCoy, 51 Woodward Building, Birmingham, Ala.

ARKANSAS STATE MEDICAL SOCIETY, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. C. G. Hinkle
Secretary: Dr. K. W. Cosgrove, 113 East Capitol Avenue, Little Rock, Ark.

CALIFORNIA MEDICAL ASSOCIATION, EYE, EAR, NOSE AND THROAT SECTION

Chairman: Dr. Robert C. McNaught
Secretary: Dr. Alfred R. Robbins, 1930 Wilshire Blvd., Los Angeles, Calif.

COLORADO OPHTHALMOLOGICAL SOCIETY

President: Dr. Fritz Nelson
Secretary: Dr. James C. Strong, Jr., 550 Metropolitan Building, Denver, Colo.
Time and Place: First Saturday of each month, October through May, University of Colorado School of Medicine, Denver

COLORADO OTOLARYNGOLOGICAL SOCIETY

President: Dr. John Bradley
Secretary: Dr. Robert C. Shattuck, 406 Republic Building, Denver, Colo.
Time and Place: First Saturday of each month, October through May, University of Colorado School of Medicine, Denver

FLORIDA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Charles C. Grace
Secretary-Treasurer: Dr. Carl S. McLemore, 1202 South Kuhl Avenue, Orlando, Fla

GEORGIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Stacy C. Howell
Secretary: Dr. Alton V. Hallum, 245 Doctors Building, Atlanta 3, Ga.

INDIANA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. E. L. Van Buskirk
Secretary-Treasurer: Dr. John R. Swan, 23 East Ohio Street, Indianapolis 4, Ind.

IOWA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Byron Merkel
Secretary: Dr. Carl A. Noe, 601-611 Highley Building, Cedar Rapids, Iowa

KANSAS STATE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. N. L. Francis
Secretary: Dr. W. D. Pitman, First National Bank Building, Pratt, Kan.

MASSACHUSETTS MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY
Chairman: Dr. Edwin B. Dunphy
Secretary: Dr. John E. McKeigue, 403 Commonwealth Avenue, Boston 15, Mass.

MICHIGAN STATE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY
Chairman: Dr. J. E. Croushore (Otolaryngology)
Co-Chairman: Dr. J. C. Gemeroy (Ophthalmology)
Secretary: Dr. R. W. Teed, 215 South Main, Ann Arbor, Mich. (Otolaryngology)
Co-Secretary: Dr. F. B. Heckert, 1105 Bank of Lansing Building, Lansing, Mich. (Ophthalmology)

MICHIGAN TRIOLOGICAL SOCIETY
President: Dr. William D. Irwin
Secretary: Dr. V. E. Cortopassi, 324 South Washington Avenue, Saginaw, Mich.
Time: Second Thursday of November, December, March, April and May

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. K. A. Phelps
Secretary-Treasurer: Dr. R. W Holmen, Lowry Medical Arts Building, St. Paul 2, Minn.
Time and Place: Second Friday of each month, November through May. Alternating, Minnesota Club, St. Paul, and Minneapolis Club, Minneapolis

MONTANA ACADEMY OF OTO-OPHTHALMOLOGY
President: Dr. Fritz D. Hurd
Secretary-Treasurer: Dr. H. L. Casebeer, Pennsylvania Building, 44 West Park Street, Butte, Mont.
Time and Place: February 22-23, 1953, Butte, Mont.

NEBRASKA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Maynard A. Wood
Secretary-Treasurer: Dr. Paul L. Peterson, 702 Sharp Building, Lincoln, Neb.

NEW JERSEY SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Henry B. Orton
Secretary: Dr. Albert F. Moriconi, 438 Hamilton Avenue, Trenton 9, N. J.

MEDICAL SOCIETY OF NEW JERSEY, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY
Chairman: Dr. Robison D. Harley
Secretary: Dr. R. F. Roh, 198 Clinton Avenue, Newark, N. J.

NEW JERSEY OPHTHALMOLOGICAL SOCIETY
President: Dr. Robison D. Harley
Secretary: Dr. Charles E. Jaeckle, 136 Evergreen Place, East Orange, N. J.

NEW MEXICO OPHTHALMOLOGIC SOCIETY
President: Dr. A. L. Schonbolz
Secretary: Dr. Howard Peck, Medical Arts Square, Albuquerque, N. M.
Time: Meetings four times a year

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. MacLean B. Leath
Secretary-Treasurer: Dr. George B. Ferguson, 1110 West Main St., Durham, N. C.
Time and Place: September 14 to 16, 1953, Francis Marion Hotel, Charleston, S. C. (Jointly with South Carolina Society of Ophthalmology and Otolaryngology)

OREGON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Richard S. Fixott
Secretary: Dr. George E. Chamberlain, 909 Medical Dental Building, Portland 5, Ore.
Time and Place: Third Tuesday each month September through May. Annual Spring Convention, March 23 to 27, 1953, Old Heathman Hotel, Portland.

PENNSYLVANIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Samuel T. Buckman
Secretary: Dr. Daniel S. DeStio, Highland Building, Pittsburgh 6, Pa.
Time: February 25, 1953

PUERTO RICO MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Luis J. Fernandez
Secretaries: Dr. Ricardo F. Fernandez, P.O. Box 2206, San Juan 10, Puerto Rico (Ophthalmology)
Dr. Carlos E. Munoz MacCormick, P.O. Box 604, San Juan, Puerto Rico (Otolaryngology)
Time and Place: Bimonthly. Puerto Rico Medical Association Building

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Clay W. Evatt
Secretary Treasurer: Dr. Roderick Macdonald, 330 East Main Street, Rock Hill, S. C.
Time and Place: September 14 to 16, 1953, Francis Marion Hotel, Charleston, S. C. (Jointly with North Carolina Eye, Ear, Nose and Throat Society)

TENNESSEE STATE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Roland H. Myers
Secretary-Treasurer: Dr. John T. Bryan, 2105 Hayes Street, Nashville, Tenn.
Time: Annually

TEXAS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. J. Charles Dickson
Secretary: Dr. Lyle Hooker, Esperson Building, Houston, Texas

VIRGINIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. A. D. Morgan
Secretary-Treasurer: Dr. G. Slaughter Fitz-Hugh, 104 East Market St., Charlottesville, Va.
Time and Place: Spring meeting, May 1953, Hot Springs, Va.

WASHINGTON STATE MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. George A. Hanson
Secretary-Treasurer: Dr. Claude K. Miller, Widby Building, Wenatchee, Wash.

WEST VIRGINIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Melvin K. McGehee
Secretary-Treasurer: Dr. Frederick C. Reel, Medical Arts Building, Charleston, W. Va.
Time and Place: May 2-3, 1953, Hot Springs, Va. Joint meeting with Virginia Society

LOCAL

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY, SECTION ON EYE, EAR, NOSE AND THROAT
Chairman: Dr. William F. Krone
Secretary: Dr. Francis J. Grant, 1224 Salem Avenue, Hillside, N. J.
Time and Place: 8:45 p.m., second Monday of the month. The Academy of Medicine, 91 Lincoln Park, South, Newark

AKRON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. C. R. Anderson
Secretary-Treasurer: Dr. A. L. Peter, 406 Akron Savings and Loan Building, Akron, Ohio
Time and Place: 6:30 p.m., first Monday in January, March, May and November. Akron City Club

ALUMNI ASSOCIATION NEW YORK EYE AND EAR INFIRMARY
President: Dr. W. T. Boland
Secretary-Treasurer: Dr. Hunter Romaine, 111 East 65th Street, New York, N. Y.

ATLANTA EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. F. Phinizy Calhoun, Jr.
Secretary-Treasurer: Dr. James T. King, 384 Peachtree Street N.E., Atlanta 3, Ga.
Time and Place: 7:30 p.m., fourth Monday of each month, October to May. Academy of Medicine

BALTIMORE CITY MEDICAL SOCIETY, OPHTHALMOLOGICAL SECTION
Chairman: Dr. M. Elliott Randolph
Secretary-Treasurer: Dr. Abraham Kremen, 2355 Eutaw Place, Baltimore 17, Md.
Time and Place: 8:00 p.m., fourth Thursday of every month October to May. Medical and Chirurgical Faculty, 1211 Cathedral St.

BIRMINGHAM EYE, EAR, NOSE AND THROAT SOCIETY
President: Each member, in alphabetical order.
Secretary: Dr. David A. McCoy, Woodward Building, Birmingham, Ala.
Time and Place: 6:30 p.m., second Tuesday of each month, September through May. Thomas Jefferson Hotel

BOSTON CITY HOSPITAL AURAL AND OPHTHALMIC ASSOCIATION
President: Dr. Joseph Nerbonne
Secretary: Dr. Benjamin Riseman, 41 Bay State Road, Boston 15, Mass.

BROOKLYN OPHTHALMOLOGICAL SOCIETY
President: Dr. Daniel K. Kravitz
Secretary-Treasurer: Dr. Louis Freimark, 256 Rochester Avenue, Brooklyn 13, N. Y.
Time and Place: 8 p.m., third Thursday in October, December, February and April. Medical Society of the County of Kings, 1313 Bedford Avenue

BUFFALO OPHTHALMOLOGIC CLUB
President: Dr. Milton A. Palmer
Secretary: Dr. Elizabeth P. Olmsted, 568 Lafayette Avenue, Buffalo 22, N. Y.
Time and Place: Second Thursday of each month, October through May. Park Lane

CENTRAL ILLINOIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. G. C. Otrich
Secretary-Treasurer: Dr. Alfred G. Schultz, 237 East State Street, Jacksonville, Ill.

CENTRAL NEW YORK EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. Donald E. Moore
Secretary-Treasurer: Dr. Lee R. Stoner, 1100 East Geness Street, Syracuse 10, N.Y.

CHATTANOOGA EYE, EAR, NOSE AND THROAT SOCIETY
Chairman and Secretary: Dr. Willard H. Steele, Jr., 552 McCallie Avenue, Chattanooga, Tenn.
Time and Place: 6:30 p.m., fourth Thursday in each month, October through May. Mountain City Club

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY
President: Dr. Arthur J. Coombs
Secretary: Dr. Lawrence J. Lawson, 636 Church Street, Evanston, Ill.

CHICAGO OPHTHALMOLOGICAL SOCIETY
President: Dr. William F. Hughes, Jr.
Secretary-Treasurer: Dr. Gail R. Soper, 636 Church Street, Evanston, Ill.
Time and Place: 7:30 p.m., third Monday of each month, October to May. Chicago-Illini Union, 715 S. Wood Street

CINCINNATI OPHTHALMOLOGIC CLUB
Presidium: Dr. Donald J. Lyle, Dr. Karl W. Ascher, Dr. Josef D. Weintraub
Secretary: Dr. Josef D. Weintraub, 715 Provident Bank Building, Seventh and Vine Streets, Cincinnati, Ohio
Time and Place: Second Wednesday night of month, November through May. University Club, Fourth and Broadway

CINCINNATI OTOLARYNGOLOGIC SOCIETY
President: Dr. Gerson Lowenthal
Secretary-Treasurer: Dr. Stephen P. Hogg, 556 Doctors' Building, Cincinnati 2, Ohio
Time and Place: 6:30 on the third Monday of each month except July, August, September and December. University Club.

CLEVELAND OPHTHALMOLOGICAL CLUB
President: Dr. Lorand V. Johnson
Secretary-Treasurer: Dr Irwin H. Stolzar, 831 National City Bank Building, Cleveland 14, Ohio
Time and Place: Second Tuesday of November, January, February and April. University Club, Cleveland, Ohio

COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION ON OPHTHALMOLOGY
Chairman: Dr. George F. J. Kelly
Clerk: Dr. M. Luther Kauffman, Medical Arts Building, Jenkintown, Pa.
Time and Place: 8:15 p.m., third Thursday of every month, October through April. College of Physicians Building

CORPUS CHRISTI EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. Rex E. House
Secretary-Treasurer: Dr. L. David Wright, Thomas Spann Clinic, 1546 South Brownlee, Corpus Christi, Texas
Time and Place: First Wednesday of each month. La Louisiane

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. W. B. Wilkinson
Secretary: Dr. Claude D. Winborn, Medical Arts Building, Dallas 1, Texas
Time and Place: First Tuesday of each month. Melrose Hotel

DES MOINES ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. J. H. McNamee
Secretary-Treasurer: Dr. H. H. Gurau, 213 Bankers Trust Building, Des Moines, Iowa
Time and Place: 6:00 p.m., fourth Monday of each month, September through May. Des Moines Club

DETROIT OPHTHALMOLOGICAL CLUB
Secretary-Treasurer: Dr. Frederick A. Lauppe, 1249 David Whitney Building, Detroit 26, Mich.

DETROIT OPHTHALMOLOGICAL SOCIETY
President: Dr. Arthur S. Hale
Secretary: Dr. Arthur P. Wilkinson, 974 Fisher Building, Detroit 2, Mich.
Time and Place: Fourth Thursday of each month, November through April, at L'Aiglon, Fisher Building

DETROIT OTO-LARYNGOLOGICAL SOCIETY
President: Dr. B. F. Glowacki
Secretary-Treasurer: Dr. John R. Birch, 1010 Maccabees Building, Detroit 2, Mich.
Time and Place: Third Wednesday of each month, September to May. Wayne County Medical Society Building

SECTION ON OPHTHALMOLOGY OF THE MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA
President: Dr. Everett S. Caldemeyer
Secretary-Treasurer: Dr. J. Spencer Dryden, 1835 Eye Street Northwest, Washington 6, D. C.

EASTERN NEW YORK EYE, EAR, NOSE AND THROAT ASSOCIATION
President: Dr. Harry Miller
Secretary-Treasurer: Dr. Frank Furlong, 713 Union Street, Schenectady, N. Y.
Time and Place: 8 p.m., first Thursday of month, October to June. Albany, Troy, Schenectady, rotating monthly

FORT WORTH EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. Hal Maxwell
Secretary: Dr. William Skokan, 1708 Medical Arts Building, Fort Worth 2, Texas
Time and Place: 6:30 p.m., first Friday of each month, except July and August. St. Joseph's Hospital

HOUSTON ACADEMY OF MEDICINE, OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SECTION
President: Dr. Lyle Hooker
Secretary: Dr. Claude C. Cody, III, 1304 Walker Avenue, Houston 2, Texas
Time: Second Thursday of each month, October through June

EYE, EAR, NOSE AND THROAT SOCIETY OF HUDSON COUNTY
President: Dr. A. V. Saradarian
Secretary-Treasurer: Dr. A. J. Magee, 921 Bergen Avenue, Jersey City 6, N. J.
Time and Place: Second Monday of each month (September through May), Jersey City Medical Center

INDIANAPOLIS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY
President: Dr. Robert W. Dearmin
Secretary-Treasurer: Dr. Lewis E. Morrison, 603 Hume Mansur Building, Indianapolis 4, Ind.
Time and Place: 6:30 p.m., second Thursday of each month, November to May. Indianapolis Athletic Club

KANSAS CITY SOCIETY OF O. O. R. L.
President: Dr. Winfred Post
Secretary: Dr. Barnard C. Trowbridge, 411 Alameda Road, Kansas City 2, Mo.
Time and Place: Third Thursday of the month, November through May. Hotel President, 14th and Baltimore

LONG BEACH EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. Edmund D. Godwin
Secretary: Dr. James V. Keipp, 110 Pine Avenue, Long Beach 2, Calif.
Time and Place: 6:00 p.m., monthly, September through May. Seaside Memorial Hospital

LOS ANGELES OPHTHALMOLOGICAL SOCIETY
President: Dr. Deane C. Hartman
Secretary: Dr. Daniel B. Esterly, 104 North Madison Avenue, Pasadena 1, Calif.
Time and Place: First Thursday of each month, September through June Los Angeles County Medical Society Building, 1925 Wilshire Boulevard

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Victor Goodhill
Secretary: Dr. Orwyn Ellis, 523 West Sixth Street, Los Angeles 14, Calif.
Time and Place: 6:00 p.m., fourth Monday of each month, September through May. Los Angeles County Medical Association Building, 1925 Wilshire Boulevard

LOUISVILLE EYE AND EAR SOCIETY
President: Dr. N. H. Burkhead
Secretary-Treasurer: Dr. A. H. Keeney, Heyburn Building, Louisville 2, Ky.
Time and Place: Dinner meetings at 6:30 p.m. on the second Thursdays of alternate months, September through May. Mayflower Hotel, 425 W. Ormsby Street

MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA, SECTION ON OPHTHALMOLOGY
President: Dr. Joseph Dessoff
Secretary-Treasurer: Dr. J. Spencer Dryden, 1835 Eye Street Northwest, Washington 6, D. C.
Time: May 5, 1953

MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA, SECTION ON OTOLARYNGOLOGY
Chairman: Dr. Irvin Feldman
Secretary: Dr. John Louzan, 1801 Eye Street N. W., Washington 6, D. C.
Time and Place: 7:00 p.m., third Tuesday in October, November, January, March and May. Army and Navy Club, 17th and Farragut Square, N.W.

MEMPHIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
Chairman: Each member, in alphabetical order
Secretary: Dr. Roland H. Myers, 1720 Exchange Building, Memphis, Tenn.
Time and Place: Second Tuesday in each month, September through May. Memphis Eye, Ear, Nose and Throat Hospital

MILWAUKEE OTO-OPHTHALMIC SOCIETY
President: Dr. Gerhard Straus
Secretary-Treasurer: Dr. Erwin E. Grossmann, 238 West Wisconsin Avenue, Milwaukee 3, Wisconsin
Time and Place: 6:00 p.m., fourth Tuesday of each month, October to May, University Club

MONTGOMERY COUNTY MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT
President: Dr. Robert A. Bruce
Secretary-Treasurer: Dr. L. N. Shroder, 144 West Fourth Street, Greenville, Ohio
Time and Place: First Tuesday of November, February, March and May. Dayton Country Club

MONTREAL OPHTHALMOLOGICAL SOCIETY
President: Dr. J. V. V. Nicholls
Secretary: Dr. R. Charbonneau, 958 Sherbrooke St. E., Montreal, Que., Canada
Time: Second Thursday of October, December, February and April

NASHVILLE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. John W. Harned, Jr.
Secretary-Treasurer: Dr. Herbert Duncan, Bennie Dillon Building, Nashville, Tenn.
Time and Place: Third Monday evening of month, October through May. James Robertson Hotel

NEW YORK ACADEMY OF MEDICINE, SECTION ON OPHTHALMOLOGY
Chairman: Dr. James W. Smith
Secretary: Dr. A. Gerard De Voe, 30 West 59th St., New York, N. Y.
Time and Place: Third Monday of the month, October through May. New York Academy of Medicine

NEW YORK SOCIETY FOR CLINICAL OPHTHALMOLOGY
President: Dr. Abraham L. Kornzweig
Secretary: Dr. Leon H. Ehrlich, 211 Central Park West, New York 24, N. Y.
Time and Place: First Monday of the month, October to May. New York Academy of Medicine

NUECES COUNTY EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. C. N. Meador
Secretary: Dr. Rex C. House, 228 Medical-Dental Building, Corpus Christi, Texas
Time and Place: 7:30 p.m., second Monday of each month, October through May. Dusty's, 1739 South Brownlee Street, Corpus Christie, Texas

OKLAHOMA CITY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. C. W. McClure
Secretary: Dr. Richard A. Clay, 416 Northwest 13th Street, Oklahoma City, Okla.
Time and Place: First Tuesday of each month. University Hospital, 800 Northeast 13th Street, Oklahoma City

OMAHA AND COUNCIL BLUFFS OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. Thomas T. Smith
Secretary-Treasurer: Dr. G. T. Alliband, 1020 Medical Arts Building, Omaha, Neb.
Time and Place: Dinner meeting third Wednesday monthly, October to May. Omaha Club

PHILADELPHIA COUNTY MEDICAL SOCIETY, EYE SECTION

Chairman: Dr. Alfred Cowan
Secretary: Dr. John W. Deichler, 301 South 21st Street, Philadelphia 3, Pa.
Time: First Thursday of each month, November through April

PHILADELPHIA LARYNGOLOGICAL SOCIETY

President: Dr. Thomas F. Furlong, Jr.
Secretary: Dr. John J. O'Keefe, 255 South 17th Street, Philadelphia 3, Pa.

PITTSBURGH ACADEMY OF MEDICINE

President: Dr. John S. Plumer
Secretary: Dr. Samuel D. Evans, 1501-2 Park Building, Pittsburgh 22, Pa.
Time and Place: 8:00 p.m., fourth Monday of each month, October through May. Pittsburgh Academy, 322 North Craig Street

PITTSBURGH OTOLOGICAL SOCIETY

President: Dr. Daniel S. DeStio
Secretary: Dr. George C. Schein, 634 Washington Road, Pittsburgh, Pa.
Time and Place: Bimonthly

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Clifton E. Benson
Secretary-Treasurer: Dr. Willard F. Goff, 432 Stimson Building, 1215 Fourth Avenue, Seattle 1, Wash.
Time and Place: Third Tuesday of each month. Medical-Dental Building, Seattle

READING EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. J. Van Dyke Quereau
Secretary: Dr. Benjamin F. Souders, 424 Walnut Street, Reading, Pa.
Time and Place: Third Wednesday of each month, September to July (dinner meetings). Wyomissing Club

RICHMOND, VIRGINIA, EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. J. Warren Montague
Secretary-Treasurer: Dr. Charles N. Romaine, 1001 West Franklin Street, Richmond 20, Va.
Time and Place: First Tuesday of January, March, May and October. Commonwealth Club

ST. LOUIS COUNTY MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Anderson C. Hilding
Secretary: Dr. James P. Tetlie, 626 Medical Arts Building, Duluth, Minn.
Time and Place: 6:00 to 8:00 p.m., preceding the monthly St. Louis County Medical Society meeting. St. Mary's Hospital, Duluth

EAR, NOSE AND THROAT CLUB OF ST. LOUIS

Chairman: Dr. Millard F. Arbuckle
Secretary: Dr. Harry N. Glick, 1504 South Grand Boulevard, St. Louis 4, Mo.
Time and Place: Third Wednesday in November, January, March and May, University Club Building

ST. LOUIS OPHTHALMIC SOCIETY

President: Dr. Edmond B. Alis
Secretary: Dr. Benjamin Milder, 539 North Grand Avenue, St. Louis 3, Mo.
Time and Place: 8:00 p.m., fourth Friday of each month, October through April, except December. Elliott Auditorium, McMillan Hospital

SAN ANTONIO SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. George H. Brunner
Secretary: Dr. Jack B. Lee, 607 New Moore Building, San Antonio, 5, Texas
Time: Second Tuesday of each month, September through May

SAN FRANCISCO COUNTY MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. W. E. Borley, 655 Sutter Street, San Francisco, Calif.
Secretary: None
Time and Place: Fourth Tuesday of each month, except July, August and December. San Francisco County Medical Society Building

SPOKANE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Phil Greene
Secretary: Dr. Cornelius E. Hagan, Jr., 508 Old National Bank Building, Spokane, Wash.
Time and Place: Fourth Tuesday of each month, September through May. Paulsen Medical and Dental Building

SUPERIOR CALIFORNIA EYE CLUB

President: Dr. Theodore Holstein
Secretary: Dr. John Berg, 436 Medico-Dental Bldg., Sacramento 14, Calif.
Time: Every third Thursday, eight months of the year

TOLEDO EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Kenneth F. Schoenrock
Secretary: Dr. L. Reed Cranmer, 316 Michigan Street, Toledo 2, Ohio.
Time and Place: Third Tuesday of each month, November through May, Toledo Club

TORONTO ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

Chairman: Dr. Joseph C. Hill
Secretary: Dr. Harold R. Sniderman, 220 Bloor Street West, Toronto 5, Ont., Canada
Time and Place: Second Monday night of November, January, February and March. Academy of Medicine, Toronto

WESTERN PENNSYLVANIA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Joseph W. Raymond
Secretary-Treasurer: Dr. F. E. Murdock, 28½ West Scribner Avenue, DuBois, Pa.
Time: Biannually. Third Thursdays of April and September

WILKES-BARRE OPHTHALMOLOGICAL SOCIETY

President: Dr. Vincent Gallizzi
Secretary: Dr. Samuel T. Buckman, 70 South Franklin Street, Wilkes-Barre, Pa.
Time and Place: Last Tuesday of each month, October through May, except December. Luzerne County Medical Society Library, 130 South Franklin Street

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE AND REHABILITATION THE AMERICAN MEDICAL ASSOCIATION

As of February 1, 1953

Audicon **Models 400 & 415**
Mfr. NATIONAL EARPHONE CO., INC.
20-22 **Shipman St.**
Newark 2, N. J.

Audiotone Model 11
Mfr. AUDIO COMPANY OF AMERICA
5305 N. Sixth St.
Phoenix, Ariz.

Audivox Model Super 67
Mfr. AUDIVOX, INC.
123 Worcester St.
Boston 18

Aurex Models L & M
Mfr. AUREX CORP.
1117 N. Franklin St.
Chicago 10

Beltone Symphonette Model
Beltone Mono-Pac Model M
Mfr. BELTONE HEARING AID CO.
1450 W. Nineteenth St.
Chicago 8

Cleartone Models 500 & 700
Cleartone Regency Model
Mfr. AMERICAN SOUND PRODUCTS, INC.
1303 S. Michigan Ave.
Chicago 5

Dahlberg Models D-1, D-3 & D-4
Dahlberg Junior Model D-2
Mfr. THE DAHLBERG CO.
Golden Valley
Minneapolis 22

Dysonic Model 1
Mfr. DYNAMIC HEARING AIDS, INC.
149 Church St.
New York 7

Electrocar Model C
Mfr. AMERICAN EARPHONE CO., INC.
10 E. Forty-third St.
New York 17

Gem Models V-35 & V-60
Mfr. THE GEM EAR PHONE CO., INC.
50 West Twenty-ninth St.
New York 1

Goldentone Models 25, 69 & 97
Mfr. JOHNSTON HEARING AID MFR. CO.
708 W. Fortieth St.
Minneapolis 8
Distr: GOLDENTONE CORPORATION
708 W. Fortieth St.
Minneapolis 8

Maico UE Atomeer
Maico Quiet Ear Models G & H
Maico Model J
Maico Top Secret Model L
Mfr. THE MAICO CO., INC.
21 N. Third St.
Minneapolis 1

Mears (Crystal & Magnetic) Aurophone Model 200
1947-Mears Aurophone Model 98
Mfr. MEARS RADIO HEARING DEVICE CORP.
1 W. Thirty-fourth St.
New York 1

Micronic Model 303
Micronic Model "Mercury"
Micronic Star Model
Mfr. AUDIVOX, INC.
123 Worcester St.
Boston 18

Microtone Classic Model T-9
Microtone Models T-10, T-612 & 45
Mfr. THE MICROTONE CO.
Ford Parkway on Mississippi
St. Paul 1

National Cub Model (C)
National Model D (Duplex)
National Standard Model (T)
National Star Model (S)
National Ultrathin Model 504
National Vanity Model 506
Mfr. NATIONAL HEARING AID LABORATORIES
106 S. Seventh St.
Philadelphia, 6

Normatone Model C
Mfr. JOHNSTON HEARING AID MFG. CO.
708 W. Fortieth St., Minneapolis 8
Distr.: NORMATONE HEARING AID CO.
22 E. Seventh St.
St. Paul 1

Otarion Model E-4
Otarion Models F-1, F-2 & F-3
Otarion Models G-2 & G-3
Mfr. OTARION, INC.
4757 N. Ravenswood Ave.
Chicago 40

Paravox Model D "Top-Twin-Tone"
Paravox Model J (Tiny-Myte)
Paravox Model XTS ('Xtra-Thin)
Paravox Model Y (YM, YC & YC-7) (Veri small)
Mfr. PARAVOX, INC.
2056 E. Fourth St.
Cleveland 15

Radioear Permo-Magnetic (Multipower)
Radioear Permo-Magnetic (Uniphone)
Radioear All-Magnetic Model 55
Radioear Model 62 Starlet
Radioear Model 72
Radioear Model 82
Mfr. E. A. MYERS & SONS, INC.
306-308 Beverly Road
Mt. Lebanon
Pittsburgh 16
Distr: RADIOEAR CORP.

Rochester Models R-1 & R-2
Mfr. ROCHESTER ACOUSTICAL LABORATORIES, INC.
117 Fourth St. S. W.
Rochester, Minn.

Silvertone Models J-92 & P-15
Mfr. JOHNSTON HEARING AID MFG. CO.
708 W. Fortieth St.
Minneapolis 8
Distr: SEARS, ROEBUCK & CO.
925 S. Homan Ave.
Chicago 7

Solo-Pak Model 99
Mfr. SOLO-PAK ELECTRONICS CORP.
Linden St.
Reading, Mass.

Sonotone Models 900, 910, 920, 925, 940 & 966
Mfr. SONOTONE CORP.
Elmsford, N. Y.

Super-Fonic Hearing Aid
Mfr. AMERICAN SOUND PRODUCTS, INC.
1303 S. Michigan Ave.
Chicago 5

Televox Model E
Mfr. TELEVOX MFG. CO.
1307 Sansom St.
Philadelphia 7

Telex Models 97, 99, 200, 300-B, 400, 500, 952 & 1700
Mfr. TELEX, INC.
Telex Park
St. Paul 1

Tonamic Model 50
Mfr. TONAMIC, INC.
12 Russell St.
Everett 49, Mass.

Tonemaster Model Royal
Tonemaster Cameo Model
Mfr. TONEMASTER, INC.
400 S. Washington St.
Peoria 2, Ill.

Unex Midget Models 95 & 110
Unex Models 200 & 230
Mfr. NICHOLS & CLARK
Hathorne, Mass.

Vacolite Models J & J-2
Mfr. VACOLITE CO.
3003 N. Henderson St.
Dallas 6

Western Electric Models 65 & 66
Mfr. AUDIVOX, INC.
123 Worcester St.
Boston 18

Zenith Miniature 75
Zenith Model Royal
Zenith Model Super-Royal
Mfr. ZENITH RADIO CORP.
5801 W. Dickens Ave.
Chicago 39

All the accepted hearing devices have vacuum tubes. Accepted hearing aids more than five years old have been omitted from this list for brevity.

TABLE HEARING AIDS

Ambco Hearing Amplifier (Table Model)
Mfr. A. M. BROOKS CO.
684 S. Bonnie Brae St.
Los Angeles 5

Aurex (Semi-Portable)
Mfr. AUREX CORP.
1117 N. Franklin St.
Chicago 10

Precision Table Hearing Aid
Mfr. PRECISION HEARING AIDS
5157 W. Grand Ave.
Chicago 39

Sonotone Professional Table Set Model 50
Mfr. SONOTONE CORP.
Elmsford, N. Y.

THE EMBRYOLOGY OF THE EYE

A motion picture in color with sound

SPONSORED BY

The American Academy of Ophthalmology and Otolaryngology

Based on material made available by the Carnegie Institution of Washington, Department of Embryology, Baltimore, Maryland, Dr. George W. Corner, director.

Produced by Sturgis-Grant Productions, Inc., with Dr. George K. Smelser, Professor of Anatomy at Columbia University College of Physicians and Surgeons, as technical advisor.

Showing time, 50 minutes

Available on one reel or in two parts on two reels
with metal containers and mailing case

Per Print — $250.00

LANTERN SLIDES *FILM STRIPS*

176 pictures have been selected by Dr. Smelser from the motion picture "The Embryology of the Eye" for use in teaching, either in conjunction with or independent of the motion picture. Legends are furnished with both slides and strips.

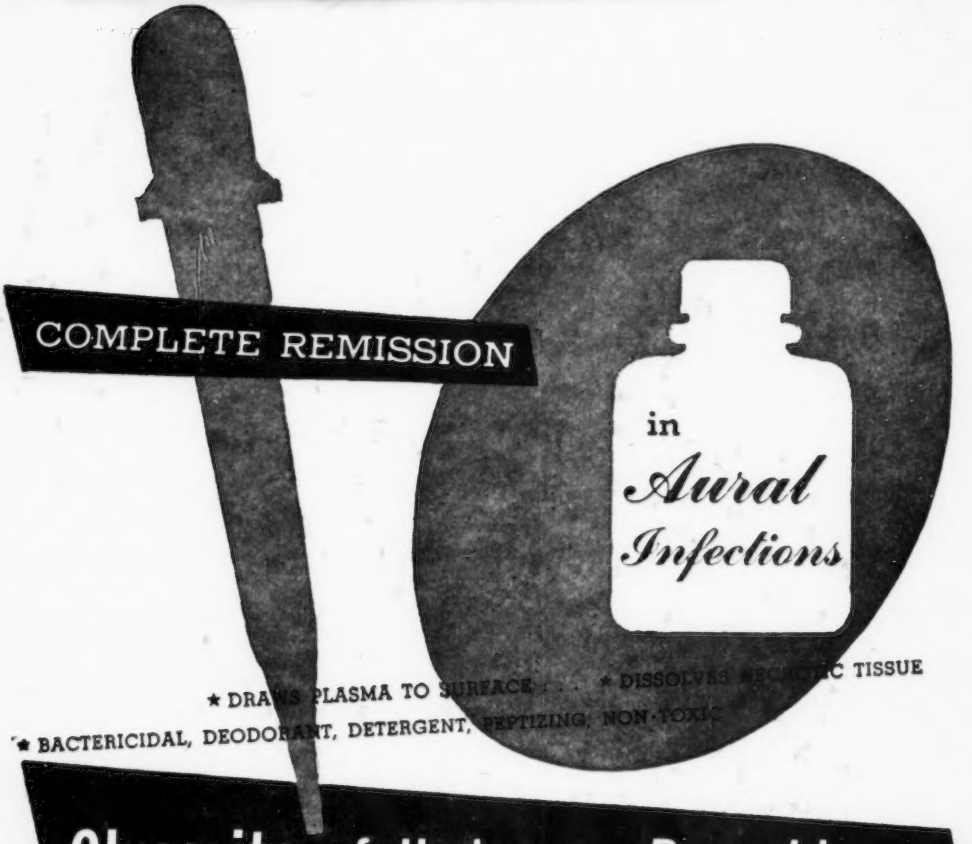
Immediate Delivery

176 Lantern Slides, 2x2, in color
Mounted in glass, with wooden filing box $125.00
Mounted in cardboard, with plastic and plywood filing box 75.00

Film Strips, 32 mm. Set of six (176 frames), in color
In metal containers 25.00

Send order to

W. L. BENEDICT, M.D.
100 First Avenue Building
Rochester, Minnesota



Glycerite of Hydrogen Peroxide *ipc*

With Carbamide

Clinical studies concerned with the use of Glycerite of Hydrogen Peroxide in the treatment of chronic purulent otitis media demonstrated seventeen of twenty-nine patients in complete remission in 14 days and the remainder by the 38th day. The patients studied presented conditions existent for periods of 2 weeks to over 40 years. Previous treatment by the usual therapeutic means, including tyrothricin or penicillin, was ineffective in all cases.

★ **BIBLIOGRAPHY**

Arch. Otolaryngol., 43:605, 1946.
E., E., N., & T. Mo., 26:27, 1947.
Laryngoscope, 56:556, 1946.
New Eng. J. Med., 234:468, 1946.
Annals of Allergy, 4:33, 1946.
J. A. Ph., A., (Sc. Ed.) 35:304, 1946.

Literature on request.

Constituents:

Hydrogen Peroxide 1.446%, Urea (Carbamide) 2.554%, 8-Hydroxyquinoline 0.1%. Dissolved and stabilized in substantially anhydrous glycerol . . . q.s. ad. 30cc.

Available on prescription in one-ounce bottle with dropper.
Administration: One-half dropperful two to four times daily.

International **PHARMACEUTICAL CORPORATION**

132 NEWBURY STREET, BOSTON 16, MASSACHUSETTS